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When it became known that Dr. M. Demerec could no longer continue to assume the responsibility for editing and publishing DIS, with the possible consequence that DIS might cease to exist, Prof. R. P. Wagner, chairman of the Committee on the Maintenance of Genetics Stocks, appointed a Subcommittee on Drosophila and Other Insects whose immediate mission was to take measures to insure the continuation of DIS as well as to look into the desirability of establishing stock centers for Drosophila and other insects.

This subcommittee, consisting of H. L. Carson, A. Chovnick, M. M. Green, W. K. Baker, G. Lefevre, E. B. Lewis (ex-officio), D. D. Miller (ex-officio), E. Novitski, M. Wheeler and R. P. Wagner, met in Chicago in December of 1959. The following decisions were reached:

1. It was agreed that the subcommittee be made a permanent one, with rotating membership. The chairman of the subcommittee should be the Drosophila member of the full committee. The heads of the Drosophila Stock Centers and Editor of DIS should be ex-officio and voting members of the subcommittee. The Editor of DIS should be given a five year term which is renewable.
2. The subcommittee suggested that Novitski serve the first five year term as Editor of DIS, in accordance with a mail poll of the subcommittee undertaken earlier by R. P. Wagner.
3. It was recommended that DIS be distributed as follows: Each independent, active Drosophila investigator should get one free copy of DIS and one free copy should go to each laboratory submitting a directory. Additional copies desired by any individual may be ordered at cost (currently $2.00).
4. It was suggested that the Editor assume the prerogative of rejecting contributions to DIS if he so wished, and that the subcommittee act as an Advisory Board to the Editor of DIS.
5. The subcommittee approved, in principle, Oster's proposal to NSF for a second Drosophila melanogaster mutant stock center (see Announcement Section, this DIS) and agreed that an effort should be made to find an investigator, trained in Drosophila taxonomy, who might be willing to assume the responsibility for maintaining a species stock center.
6. Individuals listing Drosophila stocks in DIS are advised to maintain them in the laboratory until advance notice of their being discarded is widely disseminated.

E. Novitski
Chairman, Subcommittee on
Drosophila and Other Insects

Subsequently W. K. Baker was named by R. P. Wagner to replace Novitski, who resigned both from the committee and as chairman of the subcommittee.

EDITOR'S COMMENTS

At the present time no drastic changes are contemplated, either in the format or policies of DIS, with the few minor exceptions noted below. Perhaps it is because we felt a need, nevertheless, to impress workers that some changes have been made, that we adopted the flamboyant color for the cover of this issue. The following points seem worth mentioning in detail:

The mode of reproduction: This has been changed from mimeographing to photo-offset. The former is less expensive when a small number of copies is produced, but since the number is being increased from 350 to 1,000 to provide copies for all who wish them, photo-offset is only slightly more expensive per copy. Because the cost is on a per page basis, measures have been taken to reduce the number of pages without decreasing the total content. You will notice that the size of the type has been reduced by 10% in each dimension. This makes it possible to get, in theory, 20% more material on each page. Actually the saving in space is greater than this. We have put extra columns in the stock lists and in the directory, making it possible to get from 50 to 100% more material on some of the pages. Other space-saving devices have been taken advantage of, such as the elimination of the line spacing between paragraphs found in earlier DIS's and some minor revisions of the copy sent in. There is no doubt that all this has affected the readability somewhat, but, it is hoped, not to an objectionable degree. Thus, the total amount of material in this issue is not

(continued on page 164)

AMES, IOWA: IOWA STATE UNIVERSITY

Wild Stocks

1 Ames
2 Amherst
3 Inbred
4 Iowa I
5 Iowa II
6 Oregon-R-C
7 Princeton
8 Swedish-b

Chromosome 1

9 dm/Cl, y Hw
10 ec rb cv sn
11 sc ec cv ct v f car
bb^{1}/ClB
12 y ec rb cv
13 y w sn
14 y w ec cv

Chromosome 2

15 al dp b pr c px sp
16 dp pr px/Cy pr

Chromosome 3

17 ru h st e^{s}
18 ru h th st cu sr e^{s} ca
19 se rt th/Mé

Chromosome 4

20 ci^{D}/ey^{D}

Attached-X

21 y x sc^{m}

Multichromosomal

22 al^{2} Cy lt^{3} L^{4} sp^{2}/all;
Mé Sb e^{s}/+
23 al^{2} Cy lt^{3} L^{4} sp^{2}/+,
Mé Sb e^{s}/rucuca
24 Cl Hw/Bx; al^{2} Cy lt^{3} L^{4}
Sp^{2}/+; Mé Sb e^{s}/+
25 Cy/Pm; Sb/Dfl
26 Cy; cx/cx
27 w; vg; eb; ey

AMES, IOWA: IOWA STATE UNIVERSITY
Department of Genetics
(Stocks supervised by W. F. Hollander)

Wild Stocks

1 Synthetic
2 Gilbert, Iowa

Chromosome 1 (X)

3 apr (=w^{a})
4 B
5 ct^{6} rux^{60d}
6 f
7 m
8 pn^{2}
9 pn^{2} w^{sat}
10 ras^{2}
11 ras^{2} v f
12 sc
13 sc cv v f
14 sc^{S1} B In-S w^{a}
sc^{8} (="Basc")
15 sn^{3}
16 sn^{3} g
17 v
18 w sn^{3}
19 w m
20 w f
21 w^{e} m
22 w^{sat}
23 w^{sat} rb
24 y
25 y apr
26 y w^{sat}
27 y ec rb
28 y sn^{3}

Chromosome 2

29 al
30 al dp
31 b
32 bw
33 bw^{D}
34 c px sp
35 dp
36 dp cl (?)
37 gl
38 net
39 vg

Chromosome 3

40 bx^{34e}
41 D^{3} H/Payne
42 e
43 e tx
44 fz
45 ru h th
46 se e
47 se ss ro
48 se ss ro/se C3G ro
49 ss tx
50 th
51 tx

Chromosome 4

52 ci^{D}/ey^{D}

Multichromosomal

53 ct^{6} f; bw st
54 cv B; dp
55 g; by
56 pn^{2} cv; st
57 v f; se
58 w f; cu
59 w; vg; e; ey
60 w; e
61 w; e tx
62 w^{a}; vg
63 w^{a}; ss tx
64 y w^{a}; se e
65 b pr; st
66 bw; st
67 dp cl?; e
68 vg; se
69 Cy/Pm; k?
70 k?; pol

Attached-X

71 m^{D}/+
72 w/v B
73 y/Basc (white
Mutant)
74 y/cv B
75 y/w m Bx
76 y/y sc cv v f; k?
77 y cv v f/Basc
78 y cv v f/y w f B
79 y w/B
80 y w/Basc
81 y w B/rux^{60d} ct^{6}

Translocations

82 y f Y^{L}.y^{+}/y w.Y^{S}
Y^{L}.y^{+}
83 ss bxd k e^{s}/Xa

Triploids

84 y/Basc/Basc ♂
85 y/Basc/Basc; Xa/+
86 y w/Basc/Basc ♂

Extra Y

87 y w B/Basc (lost B)
Y/y sc cv v f ♂

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

Wild Stocks

1 Oregon-R: inbreeding, generation 344 on 60129
2 Oregon-R: mass culture, extracted from $ #1 at generation 100
3 Oregon-R: mass culture, extracted from $ #1 at generation 200
4 Oregon-R: mass culture, extracted from $ #1 at generation 300
5 Oregon-R C
6 Oregon-S
7 Samarkand 204: inbred for 204 generations; mass culture since 53h4
8 Samarkand 204-30: from $ #7, inbreeding generation 30 on 60126

Chromosome 1

9 B
10 B Bx^{3}/y f:=
11 Basc (Muller-5): sc^{S1} B InS w^{a} sc^{8}
12 Basc/f.=
13 Bx
14 cm
15 cm ct^{6}
16 cm ct^{6} sn^{2}
17 cm ct^{6} sn^{2} sn/y f:=
18 cm ct^{6} sn^{3}
19 cm ct^{6} sn^{3} oc/y f:=
20 cm ct^{6} sn^{3} sn/y f:=
21 cm ct^{6} sn^{4}
22 cm ct^{6} sn^{4} oc ptg/y f:=
23 cm ct^{6} sn^{4} sn/y f:=
24 cm ct^{6} sn^{5}/y f:=
25 cm ct^{6} sn^{34e}
26 cm ct^{6} sn^{36a}/y f:=
27 cm ct^{6} sn^{36a} sn^{2} oc ptg/y f:=
28 cm sn^{2}
29 cm sn^{3}
30 cm sn^{3}; Ubx^{130} e^{s}/T(2;3) Xa
31 ct^{6}
32 ct^{6} oc/y f:=
33 ct^{n} g^{2}
34 ct^{n} oc/y f:=
35 ct^{n} sn^{3} sn^{2}/y f:=
36 f car; Ubx^{130} e^{s}/T(2;3) Xa
37 f od car
38 g
39 g sd
40 g^{2} pl
41 g^{3}
42 g^{3} sd
43 g^{50e}
44 g^{53d}
45 g^{53d} sd
46 g^{w}
47 lz^{3} m/y f:=
48 lz^{3} v/y f:=
49 oc/y f:=
50 oc ptg/y f:=
51 r^{39k} f B/In(1)AM
52 rb
53 sc^{S1} At f In49 w^{a} sc^{8}/oc ptg
54 sd
55 sn/y f:=
56 sn^{2}
57 sn^{2} oc ptg^{3}/y f:=
58 sn^{3}
59 sn^{3} g^{53d}
60 sn^{3} oc/y f:=
61 sn^{4}
62 sn^{4} oc ptg^{3}/y f:=
63 sn^{5} oc ptg/y f:=
64 sn^{34e}
65 sn^{36a}/y f:=
66 un od
67 v
68 v fw
69 v g^{53d} sd
70 v wy^{2} g^{2}
71 w
72 w/+.=
73 w sn^{5}/y f:=
74 w^{a}
75 w^{e}
76 w^{e} sn/y f:=
77 wy^{2}
78 wy^{2} g
79 wy^{2} g^{2}
80 wy^{2} g^{3}
81 wy^{2} g^{4}
82 wy^{2} g^{53d}
83 wy^{2} g^{53d} sd
84 y ct^{6} f
85 y ct^{6} ras^{2} f
86 y g^{53d} sd
87 y ras^{2} f/B.Basc
88 y sn^{50k} ras^{2}/y f:=
89 y^{2} sn^{3} ras^{4} m/B.Basc
90 y^{2} sn^{3} ras^{4} m/ClB
91 y^{2} w^{a} cv sn^{55a} v/+.=

Chromosome 2

92 a px or
93 b
94 b vg bw
95 Bl/Cy, bw^{45a} sp^{2} or^{45a}
96 Bl/Roi. bw^{45a} sp^{2} or^{45a}
97 Bl L^{2}/Cy, sp^{2}
98 cl
99 cl^{2} px sp/T (Y;2) E
100 cn bw
101 hp
102 L^{4}
103 ltd
104 net b cn bw
105 or^{49h}
106 sca/vg^{U} sca
107 sca l(2)C/SM5, Cy lt^{2} sp^{2}
108 sp^{2} or^{45a}
109 vg
110 vg^{-B}/Cy L^{4} sp^{2}
111 vg^{-D}sp^{2}/Roi, bw^{45a} sp^{2} or^{45a}
112 vg^{ni}
113 vg^{no}
114 vg^{np}
115 vg^{nw}/Roi, bw^{45a} sp^{2} or^{45a}
116 vg^{U}/Roi, bw^{45a} sp^{2} or^{45a}

Chromosome 3

117 bar-3
118 cu
119 D/Gl
120 D^{3} Sb/Payne
121 Dl^{7}/Ubx^{130} e^{s}
122 Gl Sb/LVM
123 h
124 h st
125 h th st p^{p} cu sr e^{s} (hes)
126 p^{p}
127 ri p^{p}/T(Y;2;3) F, st
128 rsd
129 ru h th st cu sr e^{s} ca (rucuca)
130 ru h st cu sr e^{s} ca
131 ru h st p^{p} ss Su^{3}-ss e^{s}
132 ru st ss ca (rusca)
133 se h
134 se ss
135 se ss Su^{3}-ss
136 ss

137 ss bx Su^{2}-ss
138 ss Su^{3}-ss
139 th
140 TM1/Ubx^{130} e^{s}
141 tx

Chromosome 4

142 ci ey^{R}
143 ey^{2}
144 pol
145 +/y f:=; vg; se

Multiple Chromosomes

146 bw; by^{46h}
147 bw; e
148 bw; e; pol
149 cn bw; e
150 vg; se^{50k}
151 vg; se^{50k}; pol
152 Cy, sp^{2}/Pm, dp b; DCxF, ru h ca/Sb sr
153 Cy, sp^{2}/Pm, dp b; H/Sb sr
154 Ubx^{130} e^{s}/T(2;3)Xa

Deficiencies

155 Df(1) g^{1}, f B/In(1)AM
* Df(2) vg^{-B} in stock 110
* Df(2) vg^{-D} in stock 111

Inversions

* In(1)AM in stocks 51, 155
* Ins(1)Basc (Muller-5) in stocks 11, 12, 87, 89
* In(1)ClB in stock 90
156 In(1)sc$^{260-22}$, sc$^{260-22}$
157 In(1)sc^{JL}; Del 24
* Ins(1)sc^{S1} 49 sc^{8} in stock 53
* In(2)bw^{V1} (Pm) in stocks 152, 153
* Ins(2)Cy in stocks 95, 97, 107, 110, 152, 153, 158
158 In(2)S^{960}/Cy, S^{e} E-S
* Ins(2L)t, (2R) Cy in stocks 96, 111, 115, 116
* Ins(3)DCxF in stocks 152, 159
* In(3)Mo, Sb sr in stocks 152, 153
* Ins(3)Payne (LVM) in stocks 120, 122
* Ins(3)TM1, Me ri sbd^{1} in stock 140
* Ins(3)Ubx^{130} in stocks 30, 36, 121, 140, 154

Translocations

* T(Y;2)E in stock 99
159 Dl^{58a17}/DCxF//T(Y;3)H^{58b25}
* T(Y;2;3)F in stock 127
* T(2;3)Xa in stocks 30, 36, 154

Triploids

160 cm ct^{6} sn^{3}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B & FM1
161 cm ct^{6} sn^{4}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B & FM1
162 cm ct^{6} sn^{36a}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B & FM1
163 ct^{n} sn^{3}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B & FM1
164 g^{53d} sd/FM1, y^{31d} sc^{8} w^{a} lz^{s} B & FM1
165 wy^{2} g^{2}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B & FM1
166 wy^{2} g^{53d} sd/FM1, y^{31d} sc^{8} w^{a} lz^{s} B & FM1

COLD SPRING HARBOR, NEW YORK: CARNEGIE INSTITUTION OF WASHINGTON

Note: Stocks are maintained primarily for use in performing the experiments outlined in Drosophila Guide, by M. Demerec and B. P. Kaufmann (7th edition, published by Carnegie Institution of Washington, 1960).

Wild Stocks*

1 Canton-S^{2}
2 Oregon-R
3 Oregon-R-EL^{2} (from East Lansing)
4 Swedish-b^{8}

*Numerical superscripts refer to successive subcultures (e.g., the eighth for Sw-b) from a single pair whose F$_{1}$ progeny were examined cytologically to determine absence of gross chromosomal aberrations.

Chromosome 1

5 B
6 bi
7 ec ct^{6} v g^{3}
8 ec ct^{6} v g^{3}/ClB
9 f
10 fw^{H}/y
11 m
12 v
13 w
14 w m f
15 w m f/ClB
16 y^{2} w^{a} cv v f B
17 y^{2} w^{a} spl
18 y^{2} w^{a} spl bi

Chromosome 2

19 bw
20 dp
21 c
22 L^{2}
23 L^{5}
24 vg

Chromosome 3

25 e
26 p^{p} by Sb^{Spi}/In(3R)C, l(3)a
27 se
28 se ss
29 st

Chromosome 4

30 ey^{2}

Multichromosomal

31 Ins(2LR)Cy/Pm^{1}, ds^{33k}; H/In(3R)C, Sb
32 Ins(2LR)SM1, al^{2} Cy sp^{2}/Pm^{2}, Ubx^{130}
e^{s}/Sb
33 y f; bw; e; ci ey^{R}
34 y; bw; e; ci ey^{R}

Inversions

35 In(1)A99b
36 In(1)sc^{S1} B InS w^{a} sc^{8} (Muller-5)
* In(1)ClB$_{3}$(8,15)
37 In(1)rst^{3}, y rst^{3} car bb
38 Ins(2LR)Cy/L; Pm
* Ins(2LR)SM1, al^{2} Cy sp^{2}/Pm^{2}; Ubx^{130}
e^{s}/Sb (32)
39 In(3L)pers
* p^{p} by Sb^{Spi}/In(3R)C, l(3)a (26)

Translocations

40 T(2;3)S^{M} Cy/vg^{nw}
41 Y^{S}X.Y^{L} , +/y^{2} su-w^{a} bb; Ore-R autosomes

Closed-X

42 X^{c2}

Attached-X

* fw^{H}/y (10)
* Y^{S}X.Y^{L}, +/y^{2} su-w^{a} w^{a} bb; Ore-R
autosomes (41)
* y f; bw; e; ci ey^{R} (33)

DeKALB, ILLINOIS: NORTHERN ILLINOIS UNIVERSITY
Department of Biological Sciences

DDT Resistant

HL1 & HL2, several strains each - sib-selected
TK1 & TK2, direct and sib-selected from mixture of resistant lines
RG, direct selected from mixture of resistant lines

DDT Sensitive

LL1 & LL2, sib-selected
Cage #1 Duplicate, descended from population that gave rise to HL1, HL2, LL1, LL2.

Several wild type stocks from DeKalb and nearby areas.

EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY
Department of Agricultural Chemistry

Wild Stocks

a1 Oregon-R (East
Lansing Y)
a2 Oregon-R-S
a3 Oregon-R-I (Iso)

Chromosome 1

b1 B
b2 B/y f:=
b3 B^{1}/y
b4 B^{1}B^{1}/y f:=
b5 bi ct^{6} g^{2}
b6 br w^{e} ec rb t^{4}/FM1,
y^{31d} sc^{8} w^{a} lz^{s} B
b7 car
b8 cm
b9 f
b10 f B/y f:=
b11 f BB/yf:=
b12 g^{2}
b13 lz/FM3, y^{31d} sc^{8} dm B l
b14 m
b15 M(1)o f/In(1)AM
b16 pa/FM4, y^{31d} sc^{8} dm B
b17 pn^{2}
b18 ras dy
b19 rb cx
b20 sc^{S1} B InS w^{a} sc^{8}
(=Muller-5=Basc)
b21 sc cv v dwx/FM6, y^{31d}
sc^{8} dm B
b22 sc ec cv ct^{6} v g^{2} f/FM3,
y^{31d} sc^{8} dm B l
b23 sc w BB^{L}, In•Y^{s} + y f:=
b24 sn^{3}
b25 t^{2} v f
b26 v
b27 w
b28 w^{e}
b29 w^{a2}
b30 w m f
b31 y
b32 y^{2} cv v f

Chromosome 2

c1 al dp b pr Bl c px sp/SM1,
al^{2} Cy sp^{2}
c2 b
c3 b cn bw
c4 b pr tk/T(Y;2)G
c5 Bla/SM5, al^{2} Cy lt^{v} sp^{2}
c6 bw
c7 bw^{D}
c8 c
c9 cn
c10 cn bw

c11 dp
c12 dp^{o} (contains vc allele)
c13 dp
c14 L^{2}
c15 M(2)173/SM5, al^{2} Cy lt^{v} sp^{2}
c16 M(2)e/Cy + T(1;2)B^{bd}/Cy
c17 mi/Pm^{2}
c18 vg

Chromosome 3

d1 bx^{34e}
d2 ca bv
d3 cd
d4 cur
d5 e^{4} wo ro
d6 e^{11}
d7 Gl Sb/LVM
d8 h
d9 M(3)1/In(3R)C, e l(3)e
d10 ru h th st cu sr e^{s} Pr ca/T(2;3)Mé
d11 se h
d12 ss
d13 ss^{a}
d14 st
d15 st c3G ca/TM1, Mé ri sbd^{1}(sp^{2})

Chromosome 4

e1 bt
e2 bt^{D}/ci^{D}
e3 Ce^{2}/spa^{Cat}
e4 ci ey^{R}
e5 ey
e6 gvl sv^{n}

Multichromosomal

f1 v;bw (1;2)
f2 y f:=;bw;e;ci ey^{R} (1;2;3;4)
f3 bw;st (2;3)
f4 Cy/Pm, ds^{33k}; H/In(3R)Mo, sr (2;3)

Attached-X

g1 y/+ (also in b3 & h2)
g2 y f:=/+ (also in b2, b4, b10, b11, b23, f2, i1 i2, i4, i6, i8,i10,i12)
* y^{2} su-w^{a} w^{a} bb:= in h1

Attached-XY

h1 Y^{s}·X InEn B y·Y^{L}/y^{2} su-w^{a} w^{a} bb:= (no free Y)
h2 Y^{s}·X InEn B y·Y^{L}/y (no free Y)
h3 Y^{s}·X InEn y·Y^{L} sc^{8} y^{+}

X with Y Arm Attached

* sc w B^{L}B In·Y^{s} in b23
i1 y v f·Y^{L}/Y^{s}#2 + y f:=/Y^{s}·Y^{s}#2
i2 y w·Y^{s}/sc·Y^{L} + y f:=/sc·Y^{L}
i3 y·Y^{s}#1
i4 y·Y^{s}#1/y f:=
i5 y·Y^{s}#2
i6 y·Y^{s}#2/y f:=
i7 y·Y^{s}#3
i8 y·Y^{s}#3/y f:=
i9 y·Y^{s}#4
i10 y·Y^{s}#4/y f:=
i11 y·Y^{L}#1
i12 y·Y^{L}#1/y f:=

Altered Y's

* sc·Y^{L} in i2
* Y^{s}·Y^{s} 2 in i1
* Y-bb in k6
* Y^{Su-V} in k7

Deficiencies

j1 Df(1)N^{8}/dl-49, y Hw m^{2} g^{4}
* Df(Y)Y-bb

Inversions

* In(1)AM in b15
k1 In(1)B^{M1} v
k2 In(1)B^{M2} f^{B27}
k3 In(1)B^{M2} v^{+}rev
* In(1)dl-49 in j1
* In(1)EN in h1, h2, h3
k4 In(1)w^{m4}
k5 In(1)w^{m4w}
k6 In(1)w^{m4w}; Df(Y)Y-bb
k7 In(1)w^{m4w}; Y^{Su-V}
* In(2L)Cy in c16, f4, l1
* In(2R)Cy in c16, f4, l1
* In(2LR)Pm in f4
* In(2LR)Pm^{2} in c17
* In(3L)P (=LVM) in d7
* In(3R)C in d9
* In(3R)Mo in f4

Translocations

l1 al T(2;3)101 sp^{2}/Cy L^{4} sp^{2}
* T(1;2)B^{bd} in c16
l2 T(1;3)04, D/ClB
l3 T(1;4)w^{ms}/w; ci ey^{R}
l4 T(1;4)w^{ms}/ci ey^{R}
* T(2;3)Mé in d10
* T(Y;2)G in c4

Stocks Co-isogenic with Oregon-R-I (a3)

b1, b2, b3, b4, b20, g1, g2, h2, i3, i4, i5, i6, i7, i8, i9, i10, i11, i12, k1, k2, k3, k4

GAINESVILLE, FLORIDA: UNIVERSITY OF FLORIDA
Department of Biology

Note: Only unusual stocks are listed.

Chromosome 3: se dn Sb/LVM

Multichromosomal

Sp/Sm5, al^{2} Cy lt^{v} sp^{2}; l(3)tr Sb/In(3LR)Ubx^{130}, Ubx^{130} e^{s}
Sp/Sm5, al^{2} Cy lt^{v} sp^{2}; l(3)tr Sb/In(3LR)Ubx^{130}, Ubx^{130} e^{s}; spa^{Cat}/ci^{D}
l(3)tr Sb/In(3LR)Ubx^{130}, Ubx^{130} e^{s}; spa^{Cat}/ci^{D}

LOS ANGELES, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

Note: 1) Stocks listed in DIS 32, p.15, have been discontinued.
2) Stocks presently maintained unchanged from DIS 32, p.44.

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY

Wild Stocks

1 Canton S
2 Canton SC (highly inbred)
3 Cockaponsett Forest, Conn.
4 IF-37, Idaho Falls, Idaho
5 NB-1, New Britain, Conn.
6 OZL, New Haven, Conn.
7 Oregon R
8 Oregon R (highly inbred)
9 Oregon K
10 Sevelen
11 Sevelen (highly inbred)
12 Swedish B
13 Swedish B (highly inbred)

Chromosome 1

14 B
15 B-reverted
16 bi
17 bi ct^{6} g^{2}
18 car
19 ct^{6}
20 dor/ClB
21 dor/FM4
22 fa
23 fu/ClB
24 fs NasA/M-5
25 g/ClB
26 g^{50e}
27 g^{50e}/y f:=
28 Hw^{49c}/M-5
29 l(1)48j/M-5
30 lz^{50e}
31 na/FM3, y^{31d} sc^{8} dm B l
32 pn^{2}
33 sc ec v g f/ClB
34 sc ec cv ct^{6} v g f/ClB, v
35 sc ec v g f
36 sc^{S1} B, In-S, w^{a} sc^{8}
37 sn^{3}/y f:=
38 sn^{3} v
39 sn^{3} v B
40 sn^{4} oc ptg^{3}/+:=
41 v
42 w
43 w ec
44 w m f
45 w spl
46 w^{a}
47 w^{a} v B
48 w^{a} v B/In(1)AM
49 w^{bf}
50 w^{bf}/FM4
51 w^{bl}
52 w^{e}
53 y sc w^{a}
54 y^{2} sc w^{a} ec/FM4, y^{31d} sc^{8} dm B(3N)
55 y^{2} sc w^{a} ec/y f:=
56 y^{2} v f
57 y^{2} w^{a} cv v f/M-5
58 y^{2} w^{a} cv sn^{55a} v +f
59 y^{2} w^{a} w/y f:=
60 w^{a} f w^{49c}/M-5

Chromosome 2

61 al
62 al b c sp^{2}
63 b
64 b cn vg
65 b vg
66 bs^{2}
67 bw
68 bw bs^{cy}
69 cn
70 cn bw
71 cn bw Kr/Pm
72 dp
73 dp bw^{a}
74 L^{2}/Cy sp^{2}
75 ltd^{37b}
76 ltd^{37b} vg
77 M(2)l^{2}/SM1, al^{2} Cy sp^{2}
78 M(2)z/In(2L)t, l(2)R
79 net al ex ds S ast/SM1, al Cy sp^{2}
80 pr
81 rc
82 sca
83 vg
84 vg c
85 vg Mt^{A} bw

Chromosome 3

86 Dfd^{r-1}
87 e
88 e^{4} wo ro
89 e^{11}
90 e^{s}
91 Gl Sb/LVM
92 Ly/D^{3}
93 Ly Sb/LVM
94 l(3)tr Ubx/TM1, Mé ri sbd^{1}
95 Mé, Ins ri sb^{1}/ru h D In-S CxF ca (h-24 Bloomington)
96 ru h th st cu sr e^{s} ca (w^{a})
97 se
98 se e
99 ss
100 ss^{a}
101 st
102 Ubx^{130} e^{s}/Xa

Chromosome 4

103 ci ey^{R}
104 ey^{2}
105 Scn/ey^{D}
106 sv^{de}/ey^{D}
107 sv^{n}
108 Cat/ci^{D}

Multichromosomal

109 ct^{45e} v; bw; e; ey^{2} (1;2;3;4)
110 g; cn(1;2)
111 v; bw (1;2)
112 v; bw; e (1;2;3)
113 v; bw; e; ey^{2} (1;2;3;4)
114 sc^{S1} B In-S w^{a} sc^{8}; SM1 Cy/Pm; Ubx/Sb (1;2;3) (H-40)
115 v; e (1;3)
116 w; e (1;3)

117 w^{a} v; e (1;3)
118 y w; ant (1;2)
119 w^{e}; cn (1;2)
120 y^{2} v f; bw (1;2)
121 bw; e (2;3)
122 bw; st (2;3)
123 cn bw; e (2;3)
124 cn; se (2;3)
125 dp; e (2;3)
126 Pm, dp b/Cy sp^{2}; Sb/CxF (ru h ca?) (2;3)
127 pr; ey^{2} (2;4)
128 e; ey^{2} (3;4)

Closed-X

129 X^{c}, y/y f:=
130 In(X^{c2})w^{vc} f/dl-49, y w lz^{s}

Deficiencies

Chromosome 1

131 Df(1)g^{1} f B/In(1)AM
132 Df(1)N^{8}/y Hw m^{2} g^{4} dl-49
133 Df(1)N^{45e}/y Hw m^{2} g^{4} dl-49
134 Df(1)N^{264-40}/y Hw m^{2} g^{4} dl-49
135 Df(1)w^{258-11}/y Hw m^{2} g^{4} dl-49
136 Df(1)w^{258-11} see number 153
137 Df(1)w^{258-42}/y Hw m^{2} g^{4} dl-49
138 Df(1)w^{258-45}/FM4, y^{31d} sc^{8} dm B
139 Df(1)w^{258-48}/y Hw m^{2} g^{4} dl-49
140 Df(1)w^{m4L} rst^{3R}/y Dp(1;3) 49^{a7}
141 Df(1)$w^{551\ 3.2}$ spl/In(1) sc^{S1} dl-49, v f B
142 Df(1)$w^{56d\ 10.1}$ spl/In(1)sc^{S1} dl-49, v f B
143 Df(1)$w^{56d\ 10.2}$/In(1)sc^{S1} dl-49, v f B
144 Df(1)w^{59k13} spl (sn^{3})/Dp $w^{V,cb}$
145 Df(1)y sc/M-5 (Vogt)

Chromosome 2

146 Df(2)bw^{5} sp^{2}/Xa
147 Df(2)Px^{2} bw sp^{2}/Cy, al^{2}, lt^{3} L^{4}
148 Df(2)vg^{B}/Cy, L^{4} sp^{2}
149 Df(2)vg^{c}/Cy, L^{4} sp^{2}
150 Df(2)vg^{D}/Cy, L^{4} sp^{2}
151 Df(2)vg^{s}/Cy, al^{2} lt^{3} L^{4} sp^{2}

Duplications

152 Dp(1;1)Co

Inversions

see 20, 21, 31, 36, 48, 77, 94, 95, etc.

Translocations

153 T(1;4)w^{VD3} (w^{258-21}), y/dl-49, y Hw m^{2} g^{4}
154 T(Y;2)C/pr cn

NORMAN, OKLAHOMA: UNIVERSITY OF OKLAHOMA

Chromosome 1

b1 ac sc pn
b2 B car & y f:=
b3 f B Bx & y f:=
b4 f BB car & y f:=
b5 v car
b6 y ac w & y f:=
b7 y sc m f^{5}
b8 y w^{a} cv v m f car & y f:=
b9 y w^{a} m car
b10 y^{2} cv v f B Bx & y f:=

Chromosome 2

c1 L^{4}
c2 vg

Chromosome 3

d1 se h

Inversions

f1 In(1)AB, B & y f:=
f2 In(1)AB, B car & y f:=
f3 In(1)AB, y cv f^{5}
f4 In(1)AB, y cv f^{5} & y f:=
f5 In_{2}(1)K
f6 In_{2}(1)K,f^{5}
f7 In_{2}(1)K, y f^{5}
f8 In(1)rst^{3}, car
f9 In(1)rst^{3}, pn f
f10 In(1)rst^{3}, w
f11 In(1)rst^{3}, y f
f12 Ins(1)sc^{S1L}, S, sc^{8R}, y sc^{S1+8} w^{a} B/sc^{8}.Y (females are XXY)

Translocation

g1 T(1;3)Dinty & y f:=

Closed-X

h1 X^{c2}, f car & y f:=

Multichromosomal

i1 y f:=; bw; e; ci ey^{R}
i2 y f:=; Y^{S}X· (FR-1), Y^{S} y w^{a} m car; net bw
i3 In(1)AM, y^{2}/Ins(1)FM6, y^{31d} dm B; Ins(2L-2R)Cy, Cy/Bl; In(3R)Vno, Vno/In(3LR)Ubx^{130}, Ubx^{130} e

X Chromosomes with a Y arm Attached

j1 Y^{S}X· (FR-1), Y^{S} y w^{a} m car & y f:=

j2 Y^{S}X· (FR-1), In(1)AB, Y^{S} y cv f^{5}
j3 Y^{S}X· (FR-1), In$_{2}$(1)K, Y^{S} y
j4 Y^{S}X· (FR-1), In$_{2}$(1)K, Y^{S} y f^{5}
j5 X·Y^{S}, f·Y^{S}/Y^{L} & sc v f.=
j6 X·Y^{L}, g^{2} B·Y^{L}/Y^{S} & y f:= (Stern)(dp T)

PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY

Note: For a description of new balancer designations appearing in this stock list, see DIS-27:57-58 for FM1, SM1 and TM1; DIS-28:77 for FM3 and FM4; DIS-29:75 for SM5; DIS-30:71 for FM6; DIS-32:81 for FMA3 and this DIS for TM3. The symbol, *, is used for cross indexing and signifies that the mutant is carried in a stock whose number is shown in parenthesis.

Wild Stocks

1 Canton-S
2 Lausanne-S
3 Oregon-R-C
4 Swedish-c
5 Urbana-S

Chromosome 1

* ac ....(162)
6 amx/FM3, y^{31d} sc^{8} dm B l
7 amx^{55}
* apr ....(144)(see w^{a})
8 Ax
* bb ....(57)
* bb^{n} ....(157)
* bb^{sc5} ....(102)
9 B
* B^{3} ....(39)
* BB ....(37)
* BB^{36b} ....(38)
* B^{i}B^{i} ....(40)
10 B Bx^{r} car/y f:=
11 Bg B/In(1)AM
12 bi ct^{6} g^{2}
13 bo
14 br
15 br w^{e} ec rb t^{4}/FM1, y^{31d}
sc^{8} w^{a} lz^{s} B
16 Bx
17 Bx^{2}
18 Bx^{3}
19 Bx^{J}
* Bx^{r} ....(10)
20 Bx^{r49k}/y f:=
21 car
* cho ....(91)
* cho^{2} ....(173)
22 cm
23 cm ct^{6}
* Co ....(170)
24 cs^{53}/y w bb.=
* ct^{6} ....(12,95,97,98)
25 ct^{n} oc/FM1, y^{31d} sc^{8} w^{a}
lz^{s} B
* cv ....(92,93,94,121)
26 cx
27 cx^{tg} t/FM1, y^{31d} sc^{8} w^{a}
lz^{s} B
28 dm/y f:=
29 dow/FM6, y^{31d} sc^{8} dm B
* dvr^{2}...............(175)
* dwx ....(92)
* dx ....(32,33)
* dxst ....
30 dy
* en^{x}-S ....(629)
31 ec
32 ec dx
33 ec dx/dl-49, y Su-Hw
Hw m^{2} g^{4}
* en^{2}-bx ....(635)
* eq ....(93)
34 Ext/FM6, y^{31d} sc^{8} dm B
35 f
36 f B/y f:=
37 f BB/y f:=
38 f BB^{36b}/y f:=
39 f B^{3}/y f:=
40 f B^{i}B^{i}/y f:=
41 f fu/y f:=
* f^{3} ....(157)
42 f^{36a}
* f^{5} ....(148)
43 fa
* fa^{n} ....(720)
44 flp
45 fo
* fu ....(41)
* fw^{34e} ....(181)
46 g^{2}
47 g^{2} pl/FM3, y^{31d} sc^{8} dm
Bl
48 g^{2} ty/y f:=
49 gg^{2}/FM6, y^{31d} sc^{8} dm B
50 gg^{3}
51 gt w^{a}
52 Hw^{49c}/FM1, y^{31d} sc^{8} w^{a}
lz^{s} B
53 if^{3}
54 kz
55 l(1)7/FM6, y^{31d} sc^{8} dm B
(nub^{2}/ +)
56 l(1)J1 sc^{J1}/l(1)J1 sc^{J1}/
De 24
57 lh B car bb/y f:=
58 lz/FM3, y^{31d} sc^{8} dm B l
59 lz^{3}/y f:=
60 lz^{34k}/y f:=
61 lz^{37h}
62 lz^{48f}/y f:=
63 lz^{BS} lz^{46f} ras^{4} v/y f:=
* lz^{D} ....(630)
* lz^{g} ....(166)
64 m
65 m^{D}/FM3, y^{31d} sc^{8} dm B l
66 M(1)n/FM6, y^{31d} sc^{8} dm
B
67 M(1)o f/In(1)AM
68 M(1)Sp/In(1)AM
69 ma-1/y f:=
70 na/y f:=
71 ny f/FM1, y^{31d} sc^{8} w^{a}
lz^{s} B (ri)
* oc ....(25)
72 od
73 pa/FM4, y^{31d} sc^{8} dm B
74 peb v
* pl ....(47)
* pn ....(99,164)
75 pn^{2}
* pn^{3} ....(96)
* ptg ....(636)
76 ptg^{2}
77 r^{9}/y f:=
* r^{12} ....(137)
78 r^{39k} f B/In(1)AM
79 ras dy
80 ras^{2}
81 ras^{3} m
82 rb
83 rb cx
84 rg
85 rst^{2}/FM1, y^{31d} sc^{8} w^{a}
lz^{s} B
86 rux/FM6, y^{31d} sc^{8} dm B
87 rux^{2}
88 s
89 sbr/y f:=
90 sc
91 sc cho
92 sc cy v dwx/FM6, y^{31d}
sc^{8} dm B
93 sc cv v eq
94 sc cv v f
95 sc ec cv ct^{6} v g^{2} f/FM3,
y^{31d} sc^{8} dm B l
96 sc pn^{3} g^{2} Bx^{2} ....
(g^{2} reverted)

97 sc z ec ct^{6}
98 sc z w^{17G2} ec ct^{6}
99 sc^{2} pn/y f:=
100 sc^{3B}
101 sc^{3-1} w/y f:=
102 sc^{5} bb^{sc5}
103 sc^{6} w^{a}
104 sc^{10} w^{a}
105 sc^{10-1}/y Hw
* sc^{D1}..............(168)
* sc^{D2}..............(169)
* sc^{J1}...............(56)
106 scp t
107 sd; (se)
108 Sh^{2}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
109 shf^{2}
* sn...............(766)
* sn^{2}...............(152)
110 sn^{3}
111 sn^{4}
112 sn^{34e}
113 sn^{36a}/y f:=
114 sp-w
115 spl
116 sta/FM3, y^{31d} sc^{8} dm B l
117 sta/y f:=
118 Su^{x}-dx dx
119 su^{2}-s v
120 su^{2}-s w^{a} cv t
121 su^{3}-s cv v f/y f:=
122 su^{S2}-v-pr v/y f:=
* su^{w}-f.............(136)
123 svr
124 svr w^{a}
125 svr^{poi}
126 sw
127 sx vb^{2} sy/In(1)AM
128 sy
129 t
130 t^{2} v f
131 t^{3}
132 tw/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
* ty ....(48)
133 un Bx^{2}/In(1)AM, ptg^{4}
134 un^{4}
135 v
136 v f su^{w}-f
137 v r^{12}
138 v^{2} fw
139 v^{36f}
140 vb
* vb^{2}...............(127)
141 vs
142 w
143 w m f
* w^{11E4}.............(186)
* w^{17G2}..............(98)
144 w^{a}...............(=apr)
145 w^{a2}
146 w^{a3}
147 w^{a4}
148 w^{bf} f^{5}
149 w^{bf2}
* w^{bf3} ....(651)
150 w^{Bwx}
151 w^{ch} wy
152 w^{co} sn^{2}
153 w^{col}
154 w^{e}
155 w^{e2}
156 w^{h}
157 w^{i} f^{3} bb^{N}
158 w^{sat}
159 w^{t} fw
160 wy
* wy^{2}...............(179)
161 y
162 y ac v
163 y ct^{K}
164 y pn
165 y sc
166 y sc lz^{g} v f/y f:=
167 y sc^{5}
168 y sc^{D1}
169 y sc^{D2}
170 y w Co/y f:=
171 y w spl
172 y^{2}
173 y^{2} cho^{2}
174 y^{2} cv v f
175 y^{2} dvr^{2} v
176 y^{2} sc w^{a} ec
177 y^{2} w^{a}
178 y^{2} w^{a} w
179 y^{2} wy^{2} g^{2} (g^{2} partly reverted?)
180 y^{2s}
181 y^{2s} fw^{34e}
182 y^{3d}/y f:=
183 y^{34c}
184 y^{td}
185 y^{v2}
186 z w^{11E4}
* z ....(97,98)

Chromosome 2

187 a px or
188 a px sp
189 ab
190 ab^{2}/T(Y;2)E
191 ab^{2} ix^{2} bw sp^{2}/Cy, dp^{Th} Bl L^{4} sp^{2}
* abb ....(383)
192 abr/XM5, al^{2} Cy lt^{v} sp^{2}
193 ad
194 al
195 al b c sp^{2}
196 al dp b bw l(2)ax/SM5, al^{2} Cy lt^{v} sp^{2}
197 al dp b pr blt bw/SM5, al^{2} Cy lt^{v} sp^{2}
198 al sp b pr Bl c px sp/SM1, al^{2} Cy sp^{2}
199 al dp b pr c px sp
200 al S ast ho/Cy, En-S
201 Alu
202 an/SM5, al^{2} Cy lt^{v} sp^{2}
203 an^{2}/Cy, bw^{v34}
204 ang
205 ant; (ro)
206 ap^{4}/SM5, al^{2} Cy lt^{v} sp^{2}
207 arch chl/SM5, al^{2} Cy lt^{v} sp^{2}
208 ast ho
209 ast^{4} dp cl
* ast^{X} ....(282)
* ay ....(311)
210 b
211 b el rd^{s} pr cn
212 b Go/Gla
213 b Go/SM5, al^{2} Cy lt^{v} sp^{2}
214 b gp
215 b j
216 b l(2)Bld pr c px sp/SM5, al^{2} Cy lt^{v} sp^{2}
217 b lt wxt bw
218 b nub pr
219 b pr tk/T(Y;2)G
220 b pr tk
221 b sf
222 b vg
* ba ....(235)
223 Bl/Cy, bw^{45a} sp^{2} or^{45a}
224 Bl/In(2LR)dp
225 Bl L^{2}/SM5, al^{2} Cy lt^{v} sp^{2}
226 Bl stw^{3}/In(2LR)dp
227 Bl stw^{48} blt tuf/SM5, al^{2} Cy lt^{v} sp^{2}
228 Bla/SM5, al^{2} Cy lt^{v} sp^{2}
229 blo
230 blt
231 bran
232 bri
233 bs^{2}
* bs^{3}..............(310)
234 bw
235 bw ba
236 bw tu
237 bw^{2b}
238 bw^{4}
239 bw^{D}
240 c
241 c wt px
242 cg c/U
243 ch
244 chl
245 chl en/SM5, al^{2} Cy lt^{v} sp^{2}
246 chl l(2)bw bw^{2b} mr^{2}/SM5, al^{2} Cy lt^{v} sp^{2}
247 chy
248 ck/SM5, al^{2} Cy lt^{v} sp^{2}

249 cl
250 cl^{2} px/T(Y;2)E
251 cn
252 cn bw
253 cn en/SM5, al^{2} Cy lt^{v} sp^{2}
254 cn^{3}/T(Y;2)C
255 cn^{35k}
256 cq
257 cr-u/Cy; (w^{e})
258 d/SM5, al^{2} Cy lt^{v} sp^{2}
259 d b/SM5, al^{2} Cy lt^{v} sp^{2}
260 da/SM1, al^{2} Cy sp^{2}
261 dil^{2} hv bw sp/SM5, al^{2} Cy lt^{v} sp^{2}
262 dke c
263 dp
264 dp^{Nov}
265 dp^{o}
266 dp^{o2}
267 dp^{Rf}/SM5, al^{2} Cy lt^{v} sp^{2}
 * dpTh............................(191)
268 dp^{tx} b/SM5, al^{2} Cy lt^{v} sp^{2}
 * dp^{v}.............................(645)
269 dp^{v2}
270 dp^{v1}/SM5, al^{2} Cy lt^{v} sp^{2}
271 ds dp
272 ds ft dp^{v2} l(2)M b pr/SM5, al^{2} Cy lt^{v} sp^{2}
273 ds S G b pr/Cy, al^{2} lt^{3} L^{4} sp^{2}
274 ds^{W}/In(2L)Cy-t, Su-S sp^{2} pr
275 ds^{38k}/Cy(2L), dp^{2} b pr
276 dsr
277 dw-24F cl/SM5, al^{2} Cy lt^{v} sp^{2}
278 dw-24F l(2)cg cg/SM5, al^{2} Cy lt^{v} sp^{2}
279 ed Su^{2}-dx
280 el
 * en ....(245)
 * esc ....(747)
281 ex
282 ex ds S^{X} ast^{X}/SM1, al^{2} Cy sp^{2}
283 fes Alu lt/SM5, al^{2} Cy lt^{v} sp^{2}
284 fj l(2)Su-H/SM5, al^{2} Cy lt^{v} sp^{2}
285 fj wt/SM5, al^{2} Cy lt^{v} sp^{2}
286 fr/Cy, sp^{2}
287 fr^{2} wt/SM5, al^{2} Cy lt^{v} sp^{2}
288 ft
 * G ....(273)
289 G^{rv}/SM5, al^{2} Cy lt^{v} sp^{2}
 * Go ....(212)
 * gp ....(214)
 * gt-4 ....(397)
 * Hia ....(418,419)
290 hk
291 hk pr
292 ho
293 hv/SM5, al^{2} Cy lt^{v} sp^{2}
294 Hx
295 hy/SM5, al^{2} Cy lt^{v} sp^{2}
296 hy a px ap/SM1, al^{2} Cy sp^{2}
 * ix ....(358)
297 j
298 J Bl/In(2L)NS
299 J^{34e}
300 kn
301 L
302 L^{2}
303 L^{4}
304 L^{5}
305 L^{G}
306 L^{K}
307 L^{r}
308 L^{si}
 * l(2)301 ....(351)
309 l(2)39a px slt sp/SM5, al^{2} Cy lt^{v} sp^{2}
310 l(2) a bs^{3}, In (2L)t/Pm ds^{33k}
311 l(2) ay b c sp/SM5, al^{2} Cy lt^{v} sp^{2}
 * l(2)C ....(381)
312 l(2) gl cn bw/SM5, al^{2} Cy lt^{v} sp^{2}
313 l(2)H L^{2}/SM5, al^{2} Cy lt^{v} sp^{2}
314 l(2)mat/SM5, al^{2} Cy lt^{v} sp^{2}
315 l(2)me/SM1 al^{2} Cy sp^{2}
316 ll^{2}
317 lm/Cy, S^{2} dp^{2} En-S
318 lt/T(Y;2)A
319 lt std/SM1, al^{2} Cy sp^{2}
320 lt stw^{3}
321 ltd
322 lw
 * lys ....(638)
323 M(2)173/SM5, al^{2} Cy lt^{v} sp^{2}
324 M(2)B/In(2L)t, l(2)B
325 M(2)B/SM5, al^{2} Cy lt^{v} sp^{2}
326 M(2)l^{2}/SM1, al^{2} Cy sp^{2}
327 M(2)p/Cy, al^{2} lt^{3} L^{4} sp^{2}
328 M(2)S3/SM1, al^{2} Cy sp^{2}
329 M(2)S5, al^{2} Cy lt^{v} sp^{2}
330 M(2)S6/SM5, al^{2} Cy lt^{v} sp^{2}
331 M(2)S7/SM5, al^{2} Cy lt^{v} sp^{2}
332 M(2)S9/SM5, al^{2} Cy lt^{v} sp^{2}
333 M(2)S11/Cy, bw^{v34}
334 M(2)S11/SM5, al^{2} Cy lt^{v} sp^{2}
335 M(2)z/SM5, al^{2} Cy lt^{v} sp^{2}
336 M(2)z Sk b/Cy(2L)dp^{2} b pr
 * Mal ....(648)
337 mi/Pm^{2}
338 mr bs^{2}/Pm, ds^{33k}
339 mr^{2}/Bld, In(2R)Cy
340 msf/SM5, al^{2} Cy lt^{v} sp^{2}
 * M-2 ....(393)
341 net
342 net al ex ds S ast shv ho rub/SM1, al^{2} Cy sp^{2}
343 net ed Su^{2}-dx
 * nub............................(218)
344 nub^{2}
345 nw^{2}/Cy-RNS
 * or ....(187)
346 pd
347 pd ll^{2} sp
349 Pfd/SM5, al^{2} Cy lt^{v} sp^{2}
350 pi/SM5, al^{2} Cy lt^{v} sp^{2}
351 pi l(2)301/SM5, al^{2} Cy lt^{v} sp^{2}
 * Pin ....(316)
352 pk cn
353 pk tuf (sp^{2}/+)
354 po vg
355 po^{2}

356 pr
357 pr cn/T(Y;2)C
358 pr cn ix/SM5, al^2 Cy lt^v sp^2
359 pr^{bw}
360 pu
361 puf
362 pw-c/SM5, al^2 Cy lt^v sp^2
363 px
364 px bl (old Berlin stock of Goldschmidt) bl = bs ?
365 px bw sp/T(Y;2)J
366 px bw mr sp/Pm, ds^{33k}
367 px slt sp
368 pys
369 Q
* rc ....(638)
370 rd/SM5, al^2 Cy lt^v sp^2
* rd^s ....(211)
371 rdo
372 rdo^2 pr
373 rh
374 rl
* rn ....(803)
375 rub
376 Ruf/Pm, ds^{33k}
377 S/Cy, En-S
378 S sp ab ltd/SM5, al^2 Cy lt^v sp^2
379 S^R/Pm, ds^{33k}
* S^X ....(282)
380 sca
381 sca l(2)C/SM5, al^2 Cy lt^v sp^2
* sf ....(221)
382 sf^2
383 shr bw^{2b} abb sp/SM5, al^2 Cy lt^v sp^2
384 shv
385 shv ho
* slt ....(309)
386 sm px/SM5, al^2 Cy lt^v sp^2
387 sm px pd/SM5, al^2 Cy lt^v sp^2
388 so
389 so^2 b cn
* sp ....(378)
390 sp^2 bs^2
391 Sp/In(2L)t, l(2)R
392 Sp/SM5, al^2 Cy lt^v sp^2
393 Sp Bl N-2/SM5, al^2 Cy lt^v sp^2
394 Sp J/In(2L)Cy-t, Su-S dp^2 pr
395 Sp J/SM5, al^2 Cy lt^v sp^2
396 Sp J L^2 Pin/SM5, al^2 Cy lt^v sp^2
397 spd gt-4/SM5, al^2 Cy lt^v sp^2
398 sple
399 spt
400 std/SM5, al^2 Cy lt^v sp^2
401 stw
402 stw^2
403 stw^3/T(Y;2)B
404 stw^5
405 stw^{48} blt tuf
406 Su-H whd l(2)Su-H/SM5, al^2 Cy lt^v sp^2
407 Su-H/Cy, pr
* Su^2-dx ....(279)
* tet ....(633)
408 Tft/Cy
409 tkd/SM5, al^2 Cy lt^v sp^2
* tk ....(220)
410 tkv
411 tri vg^{No2}/SM5, al^2 Cy lt^v sp^2
* tu ....(236)
412 tuf ltd
413 Uf
414 vg
415 vg^D/SM5, al^2 Cy lt^v sp^2
416 vg^{ni}
* vg^{No2} ....(411)
417 vg^{np}
418 vg^{nw} Hia/T(2;3)S^M Cy
419 vg^{nw} Hia/SM5, al^2 Cy lt^v sp^2
420 vg^U/Roi, bw sp or
421 vst/SM5, al^2 Cy lt^v sp^2
422 whd
423 wt
* wxt ....(217)

Chromosome 3

424 a-3
425 aa h
426 aa tu-36e
427 abd
428 app
429 as^{hg}
430 as^{hg} e^s
* At ....(792)
431 bar-3(Ives)
* Bd ....(539)
432 Bd^G/In(3R)C, l(3)a
433 bf/In(3R)C, Bs e l(3)e
* bod ....(536)
434 bp/TM1, Me ri
435 bul
436 bv
* bx ....(567)
* bx^3 ....(555)
437 bx^3 Cbx Ubx bxd pbx/Xa
438 bx^{34e}
* bx^D ....(=Ubx)
* bxd ....(437)
* by ....(548)
* c3G ....(573)
439 ca
440 ca bv
441 ca K-pn
442 ca^2
443 Cbx
444 cd
445 cmp ca/In(3R)C, e
446 cp
447 cp in ri p^p
448 cu
449 cu kar
450 cur
451 cv-c
452 cv-c sbd^2
453 cv-d
454 D/Gl
455 D^3 Sb ca^2/Payne

556 det
457 Dfd/In(3LR)Cx
458 Dfd^{r}
459 Dl H e^{s} cd/In(3R)spr, spr
460 Dl^{3}/In(3R)C, e
461 Dl^{5}/In(3R)C, l(3)a
462 Dl^{7}/In(3R)Ubx^{130}, Ubx^{130} e^{s}
463 Dl^{9}/In(3R)C, e
464 Dl^{11}/Payne, Dfd ca
465 Dl^{12}/Payne, Dfd ca
466 Dl^{13}/In(3R)C, Sb e l(3)e
467 Dl^{14}/In(3R)Cyd, Cyd
468 Dl^{x}/Payne
469 drb
470 dwh/Payne, Dfd ca
471 e^{4} wo ro
472 e^{11}
473 e^{s}
474 eg/In(3LR)Cx
475 eg^{2}/In(3LR)Cx
476 eyg
* fl ....(516)
477 fz
478 gl
479 gl^{2} e^{4}
480 gl^{3}
481 Gl Sb/LVM
482 gs
483 h
484 h^{2}
485 H/In(3R)hp, hp
486 H Pr/In(3R)C, e
487 H^{2}/Xa
488 H^{3}/In(3R)C, Sb e l(3)e
* Hn^{r}...............(492)
489 Hn^{r3} sr
490 in
491 jv
492 jv Hn^{r} h
493 jvl
* k ....(560)
* K-pn ....(441)
* kar ....(449)
494 kar^{2}
495 Ki
496 l(3)36d10/In(3LR)Cx, D
497 l(3)ac e^{s} M(3)w/LVM
498 l(3) tr Sb/In(3LR) Ubx^{130}, Ubx^{130} e^{s}
499 l(3)tr Ubx/TM1, Me ri sbd^{1}
500 ld
501 Ly/D^{3}
502 Ly Sb/LVM
503 M(3)1/In(3R)C, e l(3)e
505 M(3)36e/In(3R)C, l(3)a
506 M(3)40130/Payne, Dfd ca
507 M(3)B/In(3R)C, e l(3)e
508 M(3)B^{2}/In(3R)C, Sb e l(3)e
509 M(3)S32/T(2;3)Me
510 M(3)S34/T(2;3)Me
511 M(3)S36/T(2;3)Me
512 M(3)S37/Me
513 M(3)w/In(3R)C, e l(3)e
514 M(3)y/Me
515 ma
516 ma fl
517 mah
518 Mc/Xa
519 N-X/Xa
520 obt
521 p
522 p^{p}
523 p^{p} bx sr e^{s}
524 p^{p} cu
525 pb/In(3LR)Cx
526 pbx/Xa
527 Pc/TM1, Me ri
* Prd ....(647)
528 Pr/In(3R) C, e
529 Pr Dr/TM3, y^{+} ac^{+} ri p^{p} sep bx^{34e} e^{s}
530 Pt/Xa, ca
531 pyd
532 R Ly/In(3L)P, gm
533 ra
534 red
535 ri
536 ri bod e^{s}/Me, In(3R)C, Sb e l(3)e
537 ri p^{p}/st, T(Y;2;3)F
538 ro
539 ro Bd ca/In(3R)C, l(3)a
540 ro ra ca/T(2;3)Me
541 rs^{2}
542 rsd
* rt^{2}...............(559)
543 ru
544 ru h th st p^{p} H e^{s} ro/C(3)x, M(3)x e^{x}
C(3)x = In(3L+3R)P
545 ru h th st cu sr e^{s} ca
546 ru h th st cu sr e^{s} Pr ca/T(2;3)Me
547 ru h th st p^{p} cu sr e^{s}
548 ru^{g} jv se by
549 ry
550 ry^{2}
551 Sb/In(3LR)Ubx^{101}
552 Sb H/In(3R)C, cd
553 Sb Ubx/Xa
554 Sb^{Spi}/In(3LR)Cx
555 sbd^{2} bx^{3}
556 se
557 se app
558 se h
559 se rt^{2} th/Me
560 se ss k e^{s} ro
* sed ....(489)
561 Ser/In(3R)C, e l(3)e
562 snb
563 sr
564 sr gl
565 ss
566 ss bx
567 ss bx Su^{2}-ss
568 ss bxd k e^{s}/Xa
569 xx^{a}
570 ss^{a-B}
571 ss^{a-40a}
572 st
573 st c3G ca/TM1, Me ri sbd^{1}(sp^{2})
574 st in ri p^{p}
575 st Ki p^{p}
576 st sbd e^{s} ro ca
577 st sr e^{s} ro ca; tu 36-a
578 st sr H^{2} ca/In(3R)P^{W}, st l(3)W ca
579 st^{sp}
* su=pd ....(636)
580 su^{B}-pr/In(3R)C, e;(pr)
581 su^{2}-Hw bx bxd/TM1; Me ri sbd^{1}(sp^{2})
* Su^{2}-ss ....(567)
582 su-t(t)
583 su ve ru ve h th
584 th
585 th st cp
586 th st pb p^{p}/In(3LR)Cx
587 th st pb p^{p} cu kar su^{2}-Hw jvl ss bx sr gl/TM1, Me ri sbd^{1}
588 tra/In(3LR)Ubx^{130} (FMA3/w^{a} v)
589 tt wo
590 Tu
* tu-36e ....(426)
591 tu-h
592 tx
593 Ubx e^{4}/Payne, Dfd ca (Ubx=bx^{D})
594 ve
595 ve h th
596 ve R/In(3L)P, gm
* vo-3 ....(645)
597 W
598 W Sb/In(3LR)Cx
599 wk/Payne, Dfd ca
600 wo

Chromosome 4

601 ar/ey^{D}
602 bt
603 bt^{D}/ci^{D}
604 bt ey^{R} sv^{n}
605 Ce^{2}/spa^{Cat}
606 ci ey^{R}
607 ci gvl bt
608 ci gvl ey^{R} sv^{n}
609 ci ey^{R} sv^{n}
610 ci^{361}
611 ci^{D}/ey^{D}

612 ci^{W}
613 ci sv^{n} (±♀; w^{a}♂)
614 ey
615 ey^{2}
616 ey^{4}
* ey^{R}..........(604)
617 gvl
618 gvl ey^{R}
619 gvl sv^{n}
620 gvl ey^{R} sv^{n}
621 pol
622 spa
623 spa^{Cat}/ci^{D}
624 sv^{35a}
625 sv^{de}/ey^{D}
626 sv^{n}

Multichromosomal Stocks

627 br^{3} dxst; ed Su^{2}-dx (1;2)
628 dxst; Su-dx (1;2)
629 en^{x}-S; S/Cy (1;2)
630 lz^{D}/dl-49, m^{2} g^{4}; Cy/Pm (1;2)
631 v; bw (1;2)
632 v; In(2R)bw^{VDel}/SM1, al^{2} Cy sp^{2} (1;2)
633 sy; tet (1;2)
634 In(1)en-bx, en-bx; bx^{3} +/+ pbx (1;3)
635 y^{2} en^{2}-bx w^{bf}/FMA3, y^{2}; sbd^{2} ss
bx^{34e}/TM1, Me ri sbd^{1} (1;3)
636 ptg; px pd; su-pd (1;2;3)
637 al sp b Bl c px sp/Cy; D/C(3)X (2;3)
638 lys rc; ss (2;3)
639 y f:=; al; st; pol (1;2;3;4)
640 y f:=; bw; e; ci ey^{R} (1;2;3;4)
641 y f:=; bw; e; pol (1;2;3;4)
642 bw; st (2;3)
643 bw^{4}; st (2;3)
644 Cy/Pm, ds^{33k}; H/In(3R) Mo, sr (2;3)
645 dp^{v}; vo-3 (2;3)
646 Pm, dp b/Cy, sp^{2} Sb/D, CxF (ru h ca?)
(2;3)
647 px pd; Prd H Dp (2;3)F/Prd (2;3)
648 pr; Mal (2;4)

Attached-X

649 br ec/y^{3d}
650 f B/su^{S2}-v-pr v
651 w^{bf} M(1)36f/w^{bf3}/sn^{36a}
652 y/g^{2} ty
* y v f ....(660)
* y v f car ....(718)
653 y^{2} su-w^{a} bb/y sc^{4L} sc^{8R}
* FMA3, y^{2}/w^{a} v; (tra) ....(588)

Attached-XY

654 v f B $\overline{XY}$/y^{2} su-w^{a} w^{a} bb

Triploid

655 y^{2} sc w^{a} ec/FM4, y^{31d} sc^{8} dm B

Extra Y

656 In(1)w^{m4L}N$^{264-84R}$, y sn/FM3, y^{31d} sc^{8} dm B 1/**y ♀ dm sn ♂ (see DIS-28)**

Closed-X

657 X^{c}, y/y f:=
658 X^{c2}, cv v f/ClB
659 In(X^{c2})w^{vc}/y Hw dl-49 m^{2} g^{4} f^{5} (Catcheside) (ring stabilized)

Closed-Y

660 Y^{1c}/y w Y^{S} and y v f (Muller)

Deficiencies

Deficiencies-X

661	Df(1)259-4	Df(1)259-4/FM4, y^{31d} sc^{8} dm B
*	Df(1)sc^{8}	(699,700)
662	Df(1)260-1	Df(1)260-1/FM4, y^{31d} sc^{8} dm B
663	Df(1)B$^{263-20}$	Df(1)B$^{263-20}$/Ins(1)sc^{7}, AM, sc^{7} car
664	Df(1)bb	In(1)bb-, y sl^{2} bb-/FM4, y^{31d} sc^{8} dm B
665	Df(1)bb	In(1)bb-, y car bb-/In(1)AM
*	Df(1)bb^{1}	(677)
666	Df(1)ct$^{268-42}$	Df(1)ct$^{268-42}$, y/FM4, y^{31d} sc^{8} dm B
667	Df(1)g^{1}	Df(1)g^{1}, f B/In(1)AM
668	Df(1)N^{8}	Df(1)N^{8}/dl-49, y Hw m^{2} g^{4}

669	Df(1)N$^{264-39}$	Df(1)N$^{264-39}$ w^{ch}/FM4, y^{31d} sc^{8} dm B
670	Df(1)N$^{264-105}$	Df(1)N$^{264-105}$ (dm)/dl-49, y Hw m^{2} g^{4}
*	Df(1)rst^{2}	(85)
671	Df(1)svr	Df(1)svr, Dp(1;f)101 (Dp. het. or hom.)
672	Df(1)w$^{258-11}$	Df(1)w$^{258-11}$, y/dl-49, y Hw m^{2} g^{4}
673	Df(1)w$^{258-42}$	Df(1)w$^{258-42}$, y/dl-49, y Hw m^{2} g^{4}
674	Df(1)w$^{258-45}$	Df(1)w$^{258-45}$, y/FM4, y^{31d} sc^{8} dm B
675	Df(1)w$^{258-48}$	Df(1)w$^{258-48}$, y sc^{5} spl;Dp(1;3)w^{Vco}; y f:=
676	Df(Y)Y^{bb-}	Df(Y)Y^{bb-}, y^{2} eq
677	Df(Y)Yst	w^{e} bb^{1}/w^{e} bb^{1}; Yst and w^{e} bb^{1}; Y +; NS, px sp/l mr^{2} (Bridges)
678	Df(Y)Y"	XY', g^{2} B; Y"/y; Y(Het. In(2R)Cy) (Stern)
679	Df(2)42	Df(2)42, en/Cy, al^{2} lt^{3} L^{4} sp^{2}
680	Df(2)al	Df(2)al/Cy, En-S
681	Df(2)bw^{5}	Df(2)bw^{5} sp^{2}/Xa
682	Df(2)bw^{VDe2L}Cy^{R}	Df(2)bw^{VDe2L}Cy^{R}/Gla
683	Df(2)M33a	Df(2)M33a/Pm^{2}
684	Df(2)MB	Df(2)MB/SM1, al^{2} Cy sp^{2}
685	Df(2)MS4	Df(2)MS4/SM1, al^{2} Cy sp^{2}
686	Df(2)MS8	Df(2)MS8/SM1, al^{2} Cy sp^{2}
687	Df(2)MS10	Df(2)MS10/SM1, al^{2} Cy sp^{2}
688	Df(2)Px	Df(2)Px/Df(2)P;Dp(2;3)P/In(3R)Mo, sr; w^{e}
689	Df(2)Px^{2}	Df(2)Px^{2}, bw sp/SM1, al^{2} Cy sp^{2}
690	Df(2)rl^{10a}	Df(2)rl^{10a} lt cn/Pm, ds^{33k}
691	Df(2)S2	Df(2)S2/Cy, En-S
692	Df(2)S3	Df(2)S3;Dp(2;2)a, Cy, En-S
693	Df(2)vg^{B}	Df(2)vg^{B}/SM5, al^{2} Cy lt^{v} sp^{2}
694	Df(2)vg^{C}	Df(2)vg^{C}/Rvd
695	Df(2)vg^{C}	Df(2)vg^{C}/SM5, al^{2} Cy lt^{v} sp^{2}
*	Df(2)vg^{D}	(415)
*	Df(3)Ly	(501,502)
696	Df(3)MS31	Df(3)MS31/T(2;3)Me
697	Df(3)sbd^{105}	Df(3)sbd^{105}/Xa

Deficiencies-4

698	Df(4)M4	Df(4)M4/ey^{D}

Duplications

699	Dp(1;f)101	In(1)sc^{8}, Df(0+ac)w^{a} sc^{8} Dp(1;f)101
*	Dp(1;f)101	(671)
700	Dp(1;f)107	In(1)sc^{8}, Df(0+ac)w^{a} sc^{8}; Dp(1;f)101
701	Dp(1;f)118	In(1)sc^{8}, Df(0+ac)w^{a} sc^{8}; Dp(1;f)118
702	Dp(1;f)135	In(1)sc^{8}, Df(0+ac)w^{a} sc^{8}; Sp(1;f)135
703	Dp(1;f)X^{c2}	Dp(1;f)X^{c2}/y l(1)7/y l(1)7
704	Dp(1;f)z^{9}	Dp(1;f)z^{9}; Df(1)sc^{J4R}/y f:=
705	Dp(1;1)112	y f, Dp(1;1)112 (homozygous stock)
706	Dp(1;Y^{L})sc^{S1}	sc^{S1}·Y^{L}/y·Y^{S}; y f:=; cn bw (e/+)
707	Dp(1;3)126	v f; Dp(1;3)126/Payne, Dfd ca
708	Dp(1;3)sc^{J4}	Dp(1;3)sc^{J4}/Df(1)sc^{8}, w^{a}
709	Dp(2;2)S	Dp(2;2)S, (S ast) (S ast^{4}) net dp cl/Cy, En-S
710	Qn(1)w	Qn(1)w, (w)$_{5}$/y f:=
711	Qn(2;2)S	Qn(2;2)S, (+ast)^{5}, al ho/Cy, S^{2} En-S

Inversions

Inversions-X

712	In(1)AB	In(1)AB/y f:=
*	In(1)AM	(66,67)
713	In(1)B^{M1}	In(1)B^{M1} v (tan-like)
714	In(1)B^{M2}	In(1)B^{M2} v+ (rev)

715	In(1)B^{M2}	In(1)B^{M2} (rev) f^{B15} (rein,; mosaic)
716	In(1)B^{M2}	In(1)B^{M2} f^{B27}/ClB (Mosaic in f/f^{27})
*	In(1)bb-	(664,665)
*	In(1)Cl	(In ClB in 760,771)
717	In(1)dl-49	dl-49, ty-1
718	In(1)dl-49	dl-49, ty-2 bb^{1}/y v f car
719	In(1)dl-49	dl-49, v^{0f}
720	In(1)dl-49	dl-49, y fa^{n}
*	In(1)dl-49	(y Hw m^{2} g^{4} in 672,673)
721	Ins(1)dl-49, B^{M1}	In(1)dl-49, sc v B^{M1} (homozygous)
722	Ins(1)FM6	Ins(1)FM6, y^{31d} sc^{8} dm^{B}/y f:=
723	In(1)N$^{264-84}$	In(1)N$^{264-84}$, y/FM6, y^{31d} sc^{8} dm B
724	In(1)rst^{3}	In(1)rst^{3}, rst^{3} (homozygous)
725	In(1)rst^{3}	In(1)rst^{3}, y rst^{3} car bb
726	In(1)sc^{4}	In(1)sc^{4}, y sc^{4}
727	Ins(1)sc^{4L} sc^{8R}, y	Ins(1)sc^{4L} sc^{8R}, y
728	In(1)sc^{7}	In(1)sc^{7}, sc^{7}
729	In(1)sc^{7}	In(1)sc^{7}, sc^{7} w^{a}
730	In(1)sc^{7}, AM	Ins(1)sc^{7}, AM, sc^{7} car/FM4, y^{31d} sc^{8} dm (without B)
731	Ins(1)sc^{7}, B^{M1}	Ins(1)sc^{7}, B^{M1}, w^{43b}/y f:=
732	In(1)sc^{8}	In(1)sc^{8}, sc^{8}
733	In(1)sc^{8}	In(1)sc^{8}, sc^{8} cv y f/y f:=
734	In(1)sc^{8}	In(1)sc^{8}, y^{31d} sc^{8} w^{a}
735	Ins(1)sc^{S1L}, S, sc^{8R}	Ins(1)sc^{S1L}, S, sc^{8R}, w^{a} B (=Muller-5)
736	Ins(1)sc^{8}, dl-49	Ins(1)sc^{8}, dl-49, y^{31d} (homozygous)
*	Ins(1)sc^{8}, dl-49	(with y^{31d} w^{a} lz^{s} B in 15,25)
737	In(1)sc^{9}	In(1)sc^{9}, sc^{9} Bx f t w^{a} (homozygous)
738	In(1)sc^{J1}	In(1)sc^{J1}; Del 24
739	In(1)sc$^{260-14}$	In(1)sc$^{260-14}$, sc$^{260-14}$
740	In(1)sc$^{260-22}$	In(1)sc$^{260-22}$, sc$^{260-22}$
741	In(1)w^{m4}	In(1)w^{m4} (bb?)
742	Ins(1)y^{3PL}, S, sc^{S1R}	Ins(1)y^{3PL}, S, sc^{S1R}/y f:=; Cy/sc^{191} (Muller)
743	In(1)y^{4}	In(1)y^{4}, y^{4}

Inversions-2

744	In(2)bw^{VDe1}	In(2)bw^{VDe1}/b lt l cn mi sp
745	In(2)bw^{VDe2}	In(2)bw^{VDe2}/Rev. l

2L Inversions

746	In(2L)Cy	In(2L)Cy, al^{2} ast^{3} b pr (Cy not present)
*	In(2L)Cy	(with Cy dp^{2} b pr in 275)
*	In(2L)Cy-t	(with Su- S dp^{2} pr in 274)
*	In(2L)NS	(298)
747	In(2L)t	In(2L)t, esc c sp/SM5, al^{2} Cy lt^{v} sp^{2}
748	In(2L)t	In(2L)t, lt l L^{4} sp^{2}/Pm, ds^{33k}
*	In(2L)t	(with l(2)B in 324)

2L + 2R Inversions

749	Ins(2L+2R)Cy	Ins(2L+2R)Cy, al^{2} En-S sp^{2} (homozygous)
*	Ins(2L+2R)Cy	(in balancer as Cy; Cy S^{2} and Cy, S^{2} En-S; S^{2} and S^{2} En-S do not carry Curly mutant)
*	Ins(2L+2R)Cy, (2R)bw^{v34}, Cy	(314,315,etc.)
*	Ins(2L)Cy, (2R)NS, Cy	(333)
750	Ins(2L+2R)NS	Ns, b mr/Cy
*	Ins(2L+2R)NS	(with px sp in 677)

2LR Inversions

751	In(2LR)102, ds^{W}	In(2LR)102, ds^{W}/SM1, al^{2} Cy sp^{2}
*	In(2LR)dp	(224)

*	In(2LR)Gla	(212)
*	In(2LR)Pm	(with ds^{33k} in 310)
*	In(2LR)Pm^{2}	(337)
*	In(2LR)Rvd	(695)
*	In(2LR)U	(242)
*	In(2LR)Rev	(745)
*	Ins(2LR)SM1	(198,etc.)
*	Ins(2LR)SM5	(196,etc.)

2R Inversion

*	In(2R)Cy	(339)
*	In(2R)Mo^{K}	(792)

Inversions-3

*	In(3L)D	(D and D^{3} stocks)
752	In(3L)P$^{mot-36e}$	In(3L)P$^{mot-36e}$/R
*	In(3L)P	(with gm in 532; with Me in 559)

3L + 3R Inversions

*	Ins(3L+3R)P	(as LVM, Payne; and Payne, Dfd ca)
*	Ins(3L)P, (3R)C	with Me Sb e l(3)e (336)
753	In(3L)P, In(3R)P18	In(3L)P, In(3R)P18, Me Ubx e^{4}/In(3LR)Cx

3LR Inversions

*	In(3LR)Cx,	(474,496)
754	In(3LR)sep	In(3LR)sep, sep ri p^{p}
*	In(3LR)Ubx^{130}	(with Ubx^{130} e^{s} in 462,498)
*	Ins(3LR)TM1	see TM1 in 499, etc.
*	Ins(3LR)TM3	see TM3 in 529,etc.

3R Inversions

755	In(3R)Antp^{B}	In(3R)Antp^{B}, Antp^{B}/TM1, Me ri
*	In(3R)C	(552,486)
756	In(3R)Dl^{3}	In(3R)Dl^{B}, st Dl^{B}/In(3R)P^{W}, st l(3) W ca
*	In(3R)Cyd	(467)
*	In(3R)hp	(485)
757	In(3R)Hu	In(3R)Hu, Hu Sb^{Xpi}/Payne
758	In(3R)Mo	In(#R)Mo, sr/Xa, ca
*	In(3R)Mo	(with Sb sr in 578)
759	In(3R)P	In(3R)P^{FLA} (homozygous)
*	In(3R)P	(with st l(3)W ca in)

Translocations-1:2

760	T(1;2)Bld	T(1;2)Bld, Bld/ClB (carries In(2R)Cy)
761	T(1;2)f$^{257-5}$	T(1;2)f$^{257-5}$/In(1)AM (Demerec)
762	T(1;2)lt	T(1;2)lt/Cy (carries eq. possibly su^{3}-s)
763	T(1;2)N$^{264-10}$	T(1;2)N$^{264-10}$/FM6, y^{31d} sc^{8} dm B
764	T(1;2)scS2	T(1;2)sc^{S2}/Cy
765	T(1;2)sc^{19}	T(1;2)sc^{19}/v f:=; fes sc^{191} b pr/Cy, dpTh pr (Muller)
766	T(1;2)wy$^{274-2}$	T(1;2)wy$^{274-2}$, w^{a} sn B/In(1)AM

Translocations-1;3

767	T(1;3)263-4	T(1;3)263-4, y sc B^{1}/In(1)AM
768	T(1;3)283-3	T(1;3)283-3/w^{e} sn
769	T(1;3)"Del 143"	T(1;3)"Del 143" ru e^{s} ca/CxF, ru h ca
770	T(1;3)N$^{264-6}$	T(1;3)N$^{264-6}$y/y w dm(=N^{6})

771	T(1;3)04	T(1;3)04, D/ClB
772	T(1;3)05	T(1;3)05, D/y f:=
773	T(1;3)ras^{V}	T(1;3)ras^{V}/y f:=
*	T(1;3)sc^{J4}	(708)
774	T(1;3)sc$^{260-15}$	T(1;2)sc$^{260-15}$/dl-49, y Hw m^{2} g^{4}
775	T(1;3)v	T(1;3)v, v/FM6, y^{31d} sc^{8} dm B
776	T(1;3)w^{V}co	T(1;3)w^{V}co, v f/ClB^{36d}

Translocation-1;4

777	T(1;4)B^{S}	T(1;4)B^{S}/y f:=
778	T(1;4)N^{8a}	T(1;4)N^{8a}/FM6, y^{31d} sc^{8} dm B
779	T(1;4)sc^{8}	T(1;4)sc^{8}, B w^{a}/y f:=
780	T(1;4)w^{m5}	T(1;4)w^{m5}/ey^{D}
781	T(1;4)w^{m5}(1;3)sc^{J4}	T(1;4)w^{m5}; T(1;3)sc^{J4R}(ClB)
782	T(1;4)w$^{258-18}$	T(1;4)w$^{258-18}$, y/ci^{D}
783	T(1;4)w^{VD3} (=w$^{258-21}$)	T(1;4)w^{VD3}/Ins (1)sc^{8}, dl-49, y^{31d} w^{a} lz^{s} B
784	T(1;4)w$^{258-21}$, y w^{a}	T(1;4)w$^{258-21}$, y w^{a}/FM4, y^{31d} sc^{8} dm B

Translocations-Y;2

*	T(Y;2)A	(318)
785	T(Y;2)B	T(Y;2)B/b
*	T(Y;2)C	(357)
*	T(Y;2)E	(190)
*	T(T;2)G	(219)
*	T(Y;2)J	(305)
786	T(Y;2)rl	T(Y;2)rl, lt cn/b lt bw

Translocations-Y;2;3

*	T(Y;2;3)F	(537)

Translocation-2;3

787	T(2;3)101	al T(2;3)101 sp^{2}/Cy L^{4} sp^{2}
788	T(2;3)101	ru h T(2;3)101 e^{4} ro ca/Payne, Dfd ca
789	T(2;3)108	al T(2;3)108 c sp^{2}/Cy, al^{2} lt^{3} L^{4} sp^{2}
790	T(2;3)109	T(2;3)109 p^{P}/Payne, Dfd ca
791	T(2;3)A	Bl T(2;3)A; ru h D TA ss e^{s}/Payne
792	T(2;3)At	T(2;3)At, At/In(2R)Mo^{K}
793	T(2;3)B	al T(2;3)B sp^{2}/Cy, L^{4} sp^{2}
794	T(2;3)B	T(2;3)B; ru h D TB ss e^{s}/Payne
795	T(2;3)bw^{V4}	T(2;3)bw^{V4}/SM1, al^{2} Cy sp^{2}
796	T(2;3)bw^{V5}	T(2;3)bw^{V5}/SM5, al^{2} Cy lt^{v} sp^{2}
797	T(2;3)bw^{VDe3}	T(2;3)bw^{VDe3}, Ubx bxd/In(3LR)Cx
798	T(2;3)bw^{VDe4}	T(2;3)bw^{VDe4}/SM5, al^{2} Cy lt^{v} sp^{2}
799	T(2;3)C	T(2;3)C; ru h D TC ss e^{s}/Payne
800	T(2;3)E	T(2;3)E/SM5, al^{2} Cy lt^{v} sp^{2}
801	T(2;3)Hn	T(2;3)Hn, Hn/In(3LR)Ubx^{130}, Ubx^{130} e^{s}
*	T(2;3)Me	(512)
*	T(2;3)P	(688)
802	T(2;3)p^{Gr}	T(2;3)p^{Gr}/SM1, al^{2} Cy sp^{2}
803	T(2;3)rn	T(2;3rn/SM1, al^{2} Cy sp^{2}
804	T(2;3)Dp-s	T(2;3)Dp-s; ho/Cy, En-S(hom. viable)
805	T(2;3)S^{L}	T(2;3)S^{L}/Cy, En-S
*	T(2;3)S^{M}	(418)
806	T(2;3)Sb^{V}	T(2;3)Sb^{V}, Sb^{V}/TM3, y^{+} ac^{+} ri p^{p} sep bx^{34e} e^{s}
807	T(2;3)Xa	T(2;3)Xa/l(3)Xa R
*	T(2;3)Xa	(In 553,568)

Translocation-2;4

808 T(2;4)a	T(2;4)a/Cy, pr; ey^{2}
809 T(2;4)ast^{v}	T(2;4)ast^{v}/Cy al^{2} lt^{3} L^{4} sp^{2}
810 T(2;4)b	T(2;4)b/Cy, pr; ey^{2}
811 T(2;4)d	al sp T(2;4)d px sp/Cy pr; ey^{2}
812 T(2;4)d	T(2;4)d/Cy, pr

Translocations-3;4

813 T(3;4)a	D T(3;4)a/Me
814 T(3;4)A2	T(3;4)A2/Me, ca
815 T(3;4)A12	T(3;4)A12/Cx, D
816 T(3;4)A13	T(3;4)A13, ve ca/Me, ca
817 T(3;4)A28	T(3;4)A28, ve ca (homozygous)
818 T(3;4)c	T(3;4)c/Payne, Dfd ca
819 T(3;4)e	T(3;4)e, D/C(3)x
820 T(3;4)e	h th st T(3;4)e cu sr e^{s} ca/Payne, Dfd ca
821 T(3;4)f	T(3;4)f, h th st cu sr e^{s} ca/Payne, Dfd ca
822 T(3;4)f	T(3;4)f/Me

Transposition

823 Tp(3)bxd^{100}	Tp(3)bxd^{100}/TM1, Me ri sbd^{1}
824 Tp(3)sbd^{104}	Tp(3)sbd^{104}/Ubx^{130}
825 Tp(3)bxd^{107}	Tp(3)bxd^{107}, bx bxd^{107} sr e^{s}/bx^{34e} Mc
826 Tp(3)Vno	Tp(3)Vno/H^{2}

PHILADELPHIA, PENNSYLVANIA: THE INSTITUTE FOR CANCER RESEARCH
Division of Chemotherapy

Note: Stock list unchanged. See DIS-33. Amended list will be distributed after January 1st 1961. (Please see Notes and News announcement).

NEW YORK - 21, NEW YORK: ROCKEFELLER INSTITUTE

Note: Only unusual stocks are listed.

cn, crc/Cy
cn, crc/Pm

SALT LAKE CITY: UNIVERSITY OF UTAH
Department of Surgery, College of Medicine

1 l(1)7/dl-49 y Hw m^{2} g^{4}
2 l(1)7 e l(1)7/dl-49 y Hw
3 y lz^{3} f
4 tu^{h}
5 w^{e} sn Bl^{t}/dl-49 y Hw
6 y l(1)7/sc^{S1} B InS w^{a} sc^{8}
7 bw tu
8 st sr e^{s} ro ca; tu^{36a}
9 tu^{g}
10 tu^{48j}
11 aa tu^{36e}
12 se e^{11} tu^{49h}
13 tu^{wps}
14 bw st tu
15 f$^{257-19}$ B/In(1)AM
16 w^{bf} f$^{257-5}$
17 tu^{50d}
18 vg mt^{A} bw
19 y B$^{263-43}$ (homozygous)
20 vg bw tu
21 tu(2)49k; ma 49d
22 tu^{47}
23 tu^{50i}
24 tu^{50j}
25 tu^{51m}
26 tu^{bs}
27 tu^{1}
28 tu^{54e}
29 PW 293
30 PW 366
31 PW 472
32 PW 487
33 PW 510
34 PW 691
35 PW 851
36 PW 857
37 y^{2} sc w^{aRM}/y^{2} sc w^{a} ee
38 tu vg, y^{2}, ca
39 tu e

40 tu w
41 fu/sc^{S1} B Ins w^{a} sc^{8}
42 fu/ClB
43 Su-er tu bw; st er Su-tu
44 b(Su-er)$^{+}$bw; st er

UNIVERSITY PARK, PENNSYLVANIA: PENNSYLVANIA STATE UNIVERSITY
Department of Botany and Plant Pathology

Wild Stocks

Inbred Temperature Lines
12 lines raised at 26°C for 113 to 124 sib generations.
3 lines raised at 22°C for 95 to 102 sib generations.
9 lines raised at 18°C for 61 to 72 sib generations.
(see note concerning these lines in materials available section).

1 Ames
2 Florida
3 Iowa
4 Ives
5 Oregon
6 Princeton
7 Swede

Chromosome 1

8 B
9 Basc (Muller-5): sc^{S1} B InS w^{a} sc^{8}
10 m
11 sc cv v f
12 sn^{3}
13 w
14 y cv v f
15 y w sn
16 y w spl

Chromosome 2

17 al dp
18 al dp b pr cn px sp
19 al pr cn sp
20 dp b cn mr/Cy
21 dp pr px/Cy pr
22 ho
23 net
24 vg

Chromosome 3

25 D^{3} H/Payne
26 e^{4}
27 gl
28 h
29 ru
30 ru h st e^{s}
31 ru h th st cu sr e^{s} ca
32 S ab ltd sr/ltd
33 se
34 se ss ro
35 th cu sr e^{s} ro ca
36 tx

Multichromosomal

37 b pr st
38 bw st
39 Bx/Hw; Cy L/Pm
40 cv sp th
41 dp cl e
42 e ci
43 sc^{S1} B InS w^{a} sc^{8}; Ins SM1, al^{2} Cy sp^{2}/dp b Pm ds^{33k}; C Sb/Ubx^{130} e^{s}; pol
44 w; vg; e^{b}; ey

URBANA, ILLINOIS: UNIVERSITY OF ILLINOIS

Note: Only special stocks and those not listed in other laboratories given.

Wild Stocks

1 Original wild source unknown
2 Illinois (Urbana)
3 Illinois (lake Senachwine)

Chromosome 1

4 f B
5 f BB
6 f B^{i}
7 f B^{i}B^{i}
8 f B^{i}B
9 f B^{+}
The above six Bar series stocks are all highly inbred and isogenic.
10 y w m
11 g^{2} pl/ClB

Chromosome 2

12 al sp b Bl px sp^{2}/Cy sp^{2}

Multichromosomal

13 B; e^{11}
14 B; px; ss
15 w f; vg; e^{11}

Deficiency

16 Df(1)263-20/In(1)AM, ptg^{4}

AUSTRALIA

Adelaide, South Australia; University of Adelaide, Department of Genetics

Wild Stocks

1 Adelaide
2 Canton S

Chromosome 1

3 B
4 car v sd
5 ct v f
6 Hw f m^{2} g^{4}
7 m^{57i}
8 m^{57i} f
9 Muller-5
10 rb cx
11 sc cv v
12 v
13 v m^{57i} f
14 w
15 w^{59j}
16 w^{a}
17 w^{bl}
18 w^{sat}
19 w f
20 w^{a} B
21 y
22 y pn
23 y w
24 y w sc
25 y w^{a} cv
26 y w^{a} ct v f
27 y^{2} su-w^{a} w^{a} spl
28 $\underline{y}/lz^{55d}$
29 $\underline{y}/lz^{57j}$
30 $\underline{y}/lz^{58j}$
31 X^{c2}/sc^{S1}
32 $X.Y^{S}/Y^{LC}$

Chromosome 2

33 al
34 al sp b pr c
35 al sp b pr c px sp/Cy pr
36 b pr cn
37 b j
38 bw
39 bw vg
40 Cy L^{4}/a le cg
41 dp
42 ds b pr/Cy L
43 fj wt/Xa
44 ho
45 vg
46 vg b al

Chromosome 3

47 Bxl e^{4}/In(3R)Mo, Sb sr
Su-Sb
48 ca
49 e ro ss
50 e wo ro
51 In(3R)Hu, Hu st Sb^{SP1}/
Payne
52 Ly/D^{3}
53 ri bod e^{S}/Me, In (3R)C
Sb e l(3)e
54 ss
55 su-bxd bx bxc/In "TM"

Chromosome 4

56 ci ey^{R}
57 ey^{2}

Multichromosomal

58 bw; st
59 su-b; b pr c
60 v; st
61 v; bw
62 $\underline{y}$; Cy/Pm, ds^{33k}; H/Sb
63 y w; dp
64 e; vg
65 e; bw
66 e; dp

Melbourne, Victoria: University of Melbourne, Zoology Department

Chromosome 1

101 w
102 w^{58c}
103 w^{e-2}
104 w^{sat}
105 apr^{55k}
106 B
107 v
108 ct v f
109 bi ct^{6} g^{2}
110 bi sd Bx
111 $lz^{57j}/\underline{y}$
112 N^{8}/dl-49 y m^{2} g^{4}
113 oc ptg^{3}/ClB
114 y^{2} apr ec cv ct v f/ClB
115 y^{2} apr w^{55j} ec cv ct v f
116 sc^{S1} B InS apr sc^{8}
(Muller-5)
117 od^{54j}/Muller-5
118 sc^{S1} InS apr sc^{8}
(Muller-5 non-Bar)
119 $\underline{y}$/B
120 X^{c2}/sc^{S1}
121 "sz" + ($X.Y^{S}/Y^{Lc}$)
(Steriliser stock)
122 "sz" y (Steriliser stock)
123 $\underline{y}$ & $X.Y^{S}/Y^{Lc}$ (Steriliser
stock)
124 $\underline{y}/X^{c2}$ v f

Chromosome 2

201 el
202 ho
203 j
204 bw
205 bw ba
206 bw bs^{52d}
207 b cn c bw
208 cn bw
209 Cy cn^{2}/Pm ds^{33k}
210 fj wt/Xa T(2;3)
211 S/Cy cn^{2} L^{4} sp^{2}
212 al sp b pr c px sp/Cy pr

Chromosome 3

301 se^{51k}
302 ss
303 st
304 e^{4} ro
305 h p^{p} e^{s}
306 ru h th st cu sr e^{s} ca
(rucuca)
307 Ly/D^{3}
308 Dl^{3}/In(3R)C, e

Chromosome 4

401 ci
402 ey^{2}

Multichromosomal

501 bw; st
502 v; e^{4} ro
503 vg; se

504 T(2;3;Y)/bw; st
505 $\underline{y}$/B; bw; st
506 $\underline{y}$/v; bw; e^4 ro; ey^2
507 $\underline{y}$; Cy cn^2/Pm ds^{33k}; H/Sb
508 "sz" bw; st (Steriliser stock)
509 "sz" vg; se (Steriliser stock)

Sydney, New South Wales: The University of Sydney, Faculty of Agriculture

Wild Stock

1 Oregon-R-C

Chromosome 1

2 w
3 B
4 w f
5 y w
6 y w^e
7 sc cv v
8 ct v f
9 sc cv v f

Chromosome 2

10 dp
11 b j
12 vg pr
13 dp b j pr
14 al dp d b c px sp/Cy sp

Chromosome 3

15 e^s
16 e^{11}
17 st
18 e^4 wo ro

Chromosome 4

19 ci ey^R

Multichromosomal

20 bw; st
21 dp; e
22 v; bw

Special Stocks

23 Pr/In(3R)1
24 w f/ClB
25 $\underline{y}$/B

Sydney, New South Wales: University of Sydney, Department of Animal Husbandry

Note: Only those stocks not listed in other Australian laboratories are given.

Chromosome 1

1 pn^2
2 In rst^3

Chromosome 2

3 ex
4 net
5 sca

BELGIUM

Louvain: The University, Agricultural Institute, Laboratory of General Genetics

Wild Stocks

Abeele (Belgium)
Canton-S
Gabarros (Spain)
Swedish-B
Watou (Belgium)

Chromosome 1

Muller-5
w

Chromosome 2

bw
ho
sca
sp^2 bl^2
stw^2
vg

Chromosome 3

bv
ca
cp in ri p^p
e
jv
ri
ri e
ro
ro ve
ru h th st cu sr e^s ca
  (rucuca)
st
ve jv h th st cu sr e^s ro ca
  (vecuroca)

Multichromosomal

ca; bw
e; bw
e; vg
Muller-5; ri e
ri; stw^2
w; ri

CHILE

Santiago: Universidad de Chile, Instituto de Biología "Juan Noé", Cátedra de Biología, Zañartu 1042

Wild Stocks

1 Oregon R-C
2 Rapel
3 Santiago
4 Swedish free of inv.

Chromosome 1

5 B
6 my
7 sc^{S1} B InS w^{a} sc^{8}
8 v
9 w
10 w m f
11 y B
12 y

Chromosome 2

13 b
14 bv g
15 bw
16 dp
17 L^{2}
18 L^{2}/Cy
19 vg

Chromosome 3

20 e^{11}
21 se
22 st

Chromosome 4

23 ey^{2}

Multichromosomal

24 dp; e^{11}
25 w vg

DENMARK

Copenhagen: University of Copenhagen, Institute of Genetics

Wild Stocks

1 Oregon-R
2 Wisconsin-2
3 Wisconsin-3
4 Wisconsin-4
5 Samarkand (inbred)
6 Holte (Denmark)
7 Bøtø (Denmark)
8 Samsø (Denmark)

Chromosome 1

9 y v^{36f}
10 B
11 y^{2} su^{51c}-y ras^{2} v^{1} f
12 ras^{2} v^{1} v^{36f} m f
13 w m f
14 Muller-5
15 w
16 v (suppressible)
17 ec ct v f
18 B v
19 v^{2} fw
20 y f:= & w^{a} su-f

Chromosome 2

21 bw
22 b cn vg
23 vg
24 Cy/Pm
25 cn

Chromosome 3

26 st
27 se
28 ru h st p^{p} ss e^{s}
29 ru h th st p^{p} cu sr e^{s}
30 ru h th st cu sr e^{s} ca
31 ro
32 e
33 e ro

Chromosome 4

34 ey^{D}

Multichromosomal

35 cn bw; e
36 v; cn; st
37 vg; st
38 B; st
39 B; cn
40 B; cn bw; e
41 bw; st
42 v; bw
43 v; Cy/Pm
44 v; cn

FINLAND

Turku: University of Turku, Institute of Genetics

Wild Stocks

1 Canton-S
2 Oregon-K
3 Oregon-R-S
4 Samarkand (Inbred)

Chromosome 1

101 B
102 bi ci^{6} g^{2}
103 br w^{e} ec rb t^{4}/FM1,
y^{31d} sc^{8} w^{a} lz^{s} B
104 Bx
105 car
106 ClB/fu
107 cm ct^{6} sn^{3} ♂ & y f:= ♀
108 ct^{6}
109 cv
110 ec ct v f
111 ec dx

112 f
113 g^2 f B & y
114 In(1)dl-49, y fa^n
115 In(1)rst^3, rst^3 (homozygous)
116 In(1)sc^4, y sc^4
117 In(1)w^{m4}
118 lz/FM3, y^{31d} sc^8 dm B l
119 m
120 Muller-5
121 ras^2
122 rb
123 rb cx
124 rsc and y f:=
125 s
126 sc cv v f
127 sd (;se)
128 sn^3
129 spl
130 v
131 w
132 w^{ch} wy
133 y ac v
134 y sn^3 bb
135 z
136 x^{c2} f B & y

Chromosome Y

191 f. Y^S/Y^L
192 In(1)w^{m4} & extra Y
193 X.Y^L/Y^S & X.Y^L/X.Y^L (Neuhaus)

Chromosome 2

201 al^2 Cy, InL, lt^3/b pr,
Bl lt^3 cn^2 InCyR L^4 sp^2
202 al dp b pr c px sp
203 ap^4 vg/Cy
204 blt/Cy
205 Bl L^2/Cy
206 bw
207 cn^2 InCyR cg sp^2/InsNS px sp
208 dp^{tx} Sp cn^2/S^2 Cy cn^2
(homozygous InCyR)
209 In(2L)Cy, al^2 ast^3 b pr (Cy
not present)
210 NS, b mr/Cy
211 rl
212 stw^2
213 vg

Chromosome 3

301 bx^{34e} bxd^{121}/TM1, Mé ri
302 cu kar
303 D^3 Sb/InLP Dfd InRP
304 e
305 eg/In(3LR)Cx, D
306 Gl Sb/LVM
307 In(3R)$Antp^B$, ss^a/TM1, Mé ri sbd^1
308 In(3R)Dl^B, st Dl^B?In(3R)P^W, st l(3)W ca
309 In(3R)$P^{FLÅ}$ (homozygous)
310 jvl
311 Ly Sb/LVM
312 Me, InL Sb/ru h D InsCxF
313 Pc/T(2;3) Mé
314 R Ly/In(3L)P, gm
315 rn^2/Mé, InL
316 (ru) th st cp in ri p^p
317 se rt^2 th/Mé, InL
318 Scx/TM1, Mé ri sbd^1
319 se app
320 ss bxd k e^s/T(2;3)Xa
321 su-bxd ss^a bx^3/TM1, Mé ri sbd^1
322 th st Pc Scx p^p (ss)/TM Mé
323 tra/Mé, T23
324 Ubx^{130} e^s/TM1 Mé ri sbd^1
325 W Sb/InsCxF

Chromosome 4

401 ey
402 spa
403 sv^n

Multichromosomal

501 In(3) T(2;3)$Antp^{Yu}$/Cy; Sb
502 Cy/Pm; D/Sb (MI)
503 Cy/Pm; p^p bx sr e^s
504 Cy/Pm; ss^a/Sb
505 rl; e

Deficiencies

601 Df(2)MS-4/SM1, al^2 Cy sp^2
602 Df(2)MS-8/SM1, al^2 Cy sp^2
603 Df(2)MS-10/SM1, al^2 Cy sp^2
604 Df(2)rl^{10a} lt cn/Pm ds^{33k}

Translocations

701 T(Y;2)A/lt
702 T(Y;2)B/b c
703 T(Y;2)C/cn^3
704 Bl T(2;3)A; ru h D TA ss e^s/Payne
705 al T(2;3)101 sp^2/Cy L^4 sp^2
706 ru h T(2;3)101 e^4 ro ca/Payne, Dfd ca
707 T(2;3)Xa/Sb Ubx (and 320)
708 T(2;3)rn (several stocks with different
marker combinations) T(2;3)Mé (see 313)

FRANCE

Strasbourg: Faculté des Sciences, Laboratoire de Zoologie

Wild Stocks

Oregon R-C

Chromosome 1

B
v
w
y

Chromosome 2

cn
vg

Chromosome 3

DcxF/Dfd
e
se
se e

Chromosome 4

ey

Multichromosomal

cn; e
w; e
y; e

GERMANY

Berlin-Buch: Institut für Medizin und Biologie, Genetische Abteilung &

Berlin-Dahlem: Institut für Genetik der Freien Universität Berlin

Wild Stocks

1 Berlin wild
2 England

Chromosome 1

3 B
4 B/y w f:=
5 ClB/+
6 car
7 car bb Y; bb
8 ct
9 cv
10 dl-49, ty-l bb^{l}/y v f car:=
11 f
12 fa^{n}
13 fu^{ff}/ClB
14 gt w^{a}
15 17/dl-49, y Hw w lz^{s}
16 m
17 sc
18 sc ec ct
19 sc ec v g
20 sc ec ct v g f (Xple)
21 sc ec cv ct^{6} v s^{2} f car bb^{l}/ClB
22 sc rb cv
23 sc^{S1} InS B w sc^{8}
24 sc^{S1} InS B w^{a} sc^{8}
25 sc^{S1} InS w^{a} sc^{8}
26 spl
27 v
28 v/ClB
29 w
30 w^{a}
31 w^{bf}
32 w^{e}
33 w^{m4}
34 w sn^{3}
35 w^{co} sn^{2}
36 w^{ch} wy
37 w^{e}/yy:=
38 w^{e} bb^{l}/ClB
39 wy
40 X^{c}/yy:=
41 Xple/ClB
42 y
43 y^{303}
44 y cv v f
45 y cv v f/ClB
46 y fa wy^{2} g^{2}
47 y pn
48 y w
49 y w/ClB
50 y w bb
51 y w f:=/+
52 y v g f B/yy:=
53 yy:=/+

Chromosome 2

54 al sp
55 b cn vg
56 b pr vg
57 bw
58 bw^{pp}
59 bw cn
60 j
61 L^{2}/Cy
62 S Sp ab^{2} ltd/NS px sp
63 tu^{g}
64 vg

Chromosome 3

65 Dfd^{r-L}
66 e^{11}
67 jv se
68 p^{p}
69 ri
70 ru h st Dfd p^{p} ss e^{s}
71 ss^{a}
72 st
73 Tu

Chromosome 4

74 ar/ey^{D}
75 bt^{D}/ci^{D}
76 ci ey^{R}
77 ey^{2}

Multichromosomal

78 Bld w^{a}/w; Cy
79 cn; ss
80 Cy al^{2} lt^{3} L^{4} sp^{2}/+; C Mé Sb C/+
81 fj px sp; p^{P}/C Mé Sb C
82 se e^{11}; tu^{49h}
83 v; bw
84 vg; e^{11}
85 w; e^{11}
86 w; ss
87 y^{S1} sc^{8} InS y^{3P}; al^{2} Cy lt^{3} sp^{2}/dp b Pm^{1}; ru h D^{3} InCxF ca/Sb In(3R)
88 y f:=; bw; e; pol

Altered Y

89 Multi ♂: sc^{8}.Y/y In49 B ♂ and y f:=♀; bw^{D}
90 Multi ♀: X.Y In EN y; st
91 Sterilizer +: Y^{LC}/X.Y^{S}♂ and X.Y^{S}♀
92 Sterilizer cn bw; e: Y^{LC}/X.Y^{S}; cn bw; e ♀

Deficiencies - 4

93 Df(4)M4/ey^{D}

Heidelberg: Zoologisches Institut der Universität

Wild Stocks

Canton-S
Oregon-R-C

Chromosome 1

B
g^{2} pl/FM3
m
w
w^{co} sn^{2}
w^{e}
y
X^{c2}, cv v f/ClB

Chromosome 2

al
b
bw
cn
cn bw
vg

Chromosome 3

e^{11}
Ly/D^{3}
se

Karlsruhe: Institut für Strahlenbiologie, Kernforschungszentrum Karlsruhe

Wild Stocks

Berlin wild

Chromosome 1

B
In-49 ty-1 bb^{1}
lz^{3}
sc t^{2} v f
sc^{S1} B In-S w^{a} sc^{8} (M-5)
y
w

Chromosome 2

b cn vg
cn bw
dp
ho
vg

Chromosome 3

e

Multichromosomal

y sc^{S1} In 49 sc^{8}; bw; st

Attached-X

y f
y v f car

Marburg/Lahn: Zoologisches Institut der Philipps-Universität

Note: Only stocks not generally available in this country are listed.

Chromosome 1

In(1)N^{264-84}, y/FM6, y^{31d} sc^{8} dm B
In(1)rst^{3}, w
In(1)rst^{3}, w m f/M-5
In(1)w^{m4}; Y's extra
In(1)w^{m4}; Cy/En-Var 7
In(1)X^{c2}, w^{vc}
y sc^{S1} B, InS, w^{a} sc^{8}/v car (inbred)
y^{Si}, InS, y^{3P}/w dow 1 (inbred)
y^{2} sc^{S1} B, In49 v, w^{a} sc^{8}/y v f.Y^{L}
y^{2} sc^{S1} B, In49 v, w^{a} sc^{8}/y w.Y^{S}
y ac w & y f:=
y w spl
y z^{a}
sc z^{1}
sc z^{m}
z

Translocations

T(1;3)N^{264-6}, y/y w dm
T(1;4)w^{m5}/w; ci ey^{R}
T(1;4)w^{258-18}, y/y w dm
T(1;4)w^{258-21}/y w spl

Mariensee: Max-Planck-Institut für Tierzucht und Tierernährung

Wild Stocks

Berlin
Oregon-R
Canton-S

Chromosome 1

B
car
y f x lz^{34k}
lz^{37h}
lz^3 x y f:=
ras^2
v
ma-l
ma-l/y f:=
w
w^a
w^{bf}
w^{Bwx}
w^{ch} wy
w^{co} sn^2
w^{col}
w^h
w^{sat}
w^t fw
sc ec ct^6 v g^2 f
y fa wy^2 g^2
y pn

Chromosome 2

b cn vg
cl
S/Cy, E-S
lt/stw^3
ltd
pd
po^2
pr
rub
cn

Chromosome 1+2

$lz^D/49$ (m^2 g^4); Cy/Pm
ma-l; cn
v; ry^2

Chromosome 3

gl
gl^3
ru
kar^2
p^p
ra
ro
rs^2
ss^{A-B}
st
Dfd^{r-L}
ri ss
mwh ri ro
mah
ma
ca
ry^1
ry^2
rb
cu, kar
se
jv, Hn
ri
ss
L/D
red
red e

Chromosome 2+3

cu; ry^2
(Sb; Ubx^{130})/Xa
(Cy SM1; Ubx^{130})/Xa

Chromosome 4

ey

Chromosome 1,2,3,4

y f; bw; e; ci ey^R

Tübingen: Max-Planck-Institut für Biologie

Wild Stocks

1 Sevelen (Zürich)

Chromosome 1

2 B
3 e
4 sc^{S1} B InS w^a sc^8
5 sc^8 bb w^a
6 w
7 $\underline{y}$
8 $\underline{y}$/w
9 y sc^4 B v^{41b}/y w In49 lz^s
10 y rst^3, In car bb
11 y v f

Chromosome 3

12 D tra ca/InLP Dfd InRP ca
13 tra/Me, T 23

Multiple Chromosomes

14 (X,Y,3) sc^8Y/X.Y InEN y; ru h D InsCxF/ru tra p
15 (X,Y,3) X.Y InEN In49; st (no free Y)

Closed-X

16 In(1)X^{c2}, w^{vc}
17 sc^8Y/X^{c2}, y v & y f:=

Attached-XY

18 v f B, X-Y/$\underline{y^2 su\text{-}w^a\ w^a\ bb}$

Altered Y

19 Y^{lc}/X.Y^s("sz +")
20 Y^{lc}/w.Y^s("sz w")

Extra Y

21 In(1)w^{m4I}N$^{264-84R}$, y sn/FM3, $y^{31d}sc^{8}$dm B l/y ♀ dm sn ♂ (Pasadena 638, DIS-31)

Deficiencies

22 Df(1)bb In(1)bb^{-}, y sl^{2} bb^{-}/FM4, y^{31d} sc^{8} dm B
23 Df(Y)Y^{bb-} Df(Y)Y^{bb-}, y^{1S} eq
24 Df(Y)Y^{st} w^{e} bb^{1}/w^{e} bb^{1}; Y^{st} & w^{e} bb^{1}/Y^{+}; NS, px sp/l mr^{2} (Bridges)

Duplications

25 Dp(1;f)101 In(1)sc^{8}, Df(o+ ac)w^{a} sc^{8} Dp(1;f)101

Triploid

26 y^{2} sc w^{a} ec/FM4, y^{31d} sc^{8} dm B

GHANA

Legon, Accra: University College of Glana, Department of Zoology

Wild Stocks

1 Oregon

Chromosome 1

2 BB
3 ma-1
4 N^{8}/dl-49 y Hw m^{2} g^{4}
5 w
6 w^{a} m
7 y m car

Chromosome 2

8 cn bw
9 vg

Chromosome 3

10 Mc/Sb
11 ry
12 st
13 ve ss^{a}

Multichromosomal

14 cu ey

GREAT BRITAIN

Birmingham, England: The University, Department of Genetics

Collected Stocks: 1-9

Massed after inbreeding: 10-15

Inbred for ca. 15-600 generations

16-25

Chromosome 1

26 ec ct s car/Muller-5
27 sc cv v f/Muller-5
28 w m B
29 w^{a}
30 w^{a} v
31 y v f
32 y w

Chromosome 2

33 b
34 b cn
35 b cn c
36 b cn vg
37 b pr vg
38 b vg
39 c
40 Cy/Bl L^{4}
41 L^{4}

Chromosome 3

42 cu
43 st
44 st p^{p}

Chromosome 4

45 ey^{2}

Multichromosomal

46 cn; st
47 v; bw
48 vg; st p^{p}
49 vg; st p^{p}; ey^{2}
50 w; vg; e

Rearrangements

51 Me Sb e/H e
52 Xa
53 Cy
54 ClB/+; Cy
55 Muller-5/+; Cy L/Pm; Sb/H

Attached-X

56 y x w
57 y x sc ec cv ct v f car

Chromosome Y

58 X.Y^{L}/Y^{S}

Edinburgh, Scotland: Institute of Animal Genetics

Wild Stocks

Sao Paulo
Renfrew
Oregon-K
Kaduna
Pacific

Inbred lines

Edinburgh
Oregon-R
Pobla de Lillet
Gabarros

Chromosome 1

1 B
2 ClB/fu
3 ClB/sc v f car
4 ClB/w sn
5 ct v g
6 ec dx
7 f
8 fa
9 fwd
10 lz^3/y f:=
11 m
12 ras^2 f
13 sc w ec cv ct
14 sc^{S1} B InS w^a sc^8
15 sc^{S1} InS w^a sc^8
16 sn^3
17 sn^{36a}/y f:=
18 spl
19 v
20 v g f
21 w
22 w m
23 w sn
24 w sn B
y sc^{S1} In49 sc^8
26 y w
27 y w sn

Chromosome 2

1 ab b e sp^2
2 al sp b pr c px sp
3 ap^4/Rvd
4 ap^4/SM5 al^2 Cy lt^v sp^2
5 b cn vg
6 bw
7 bw vg
8 cg c/U
9 ch
10 cn
11 Cy/Bl L^2
12 d/SM5, al^2 Cy lt^v sp^2
13 d b/SM5, al^2 Cy lt^v sp^2
14 dp
15 dp b cn bw
16 dp cn bn
17 dp sp
18 ds S G b pr/Cy al^2 lt^3 L^4 sp^2
19 ds^W/In(2L)Cy-t, Su-S dp^2 pr
20 ds^{38k}/Cy(2L), dp^2 b pr
21 ex^2 sp/SM5, al^2 Cy lt^v sp^2
22 net
23 pr
24 px bw mr sp/Pm, ds^{33k}
25 S/Cy, En-S
26 sp^2 bs^2
27 stw
28 stw^{48} blt tuf
29 tuf ltd
30 vg

Chromosome 3

1 app
2 Bd^G/In(3R)C,
3 bv
4 ca
5 ca bv
6 cu
7 cv-c
8 e
9 gl^2 e^4
10 Gl Sb/D In CxF.
11 h
12 ma
13 Pc/TM1, Mé ri Pdr..(593)
14 ri
15 ru h th st cu sr e^s ca
16 ru h th st cu sr e^s Pr ca/T(2;3)Mé
17 ru^g jv se by
18 se app
19 se ss k e^5 ro
20 st
21 th st p^b p^P/In(3LR)C
22 tx

Chromosome 4

1 ey^2
2 M$_4$/ey^D

Attached X

1 lz^3 & y f:=
2 sc ct v f car & y v f:=
3 sc^{S1} B InS w^a sc^8 & y v f:=
4 w & y w f:=
5 w lz In49 & sc^{S1} B InS w^a sc^8:=
6 y sc^{S1} B In49 sc^8 & y f:=

Y Chromosome

1 f.Y^S/sc.Y^L & y w f:=
2 f.Y^S/Y^L
3 sc^8.Y/y B & y f:=
4 sc^{V1}.Y^8/y v f.Y^L & y f:=
5 X.Y^L/Y^S
6 Y^L/X.Y^S & X.Y^S
7 y v f.Y^L/Y^S & f:=

Multichromosomal

1 bw; e
2 bw; st
3 cu bw; e
4 Cy/Bl L^2; D/LVM
5 Cy/Pm; Mé/H
6 dp; e
7 sc^{S1} InS w^a sc^8; vg; e
8 sc^{S1} InS w^a sc^8; Cy/Bl L^2
9 sc^{S1} & y:= bw; e; ey
10 v; bw^{va}/Bl L^2
11 vg; e
12 w; bw
13 y Sc^{S1} In49 sc^8; Cy/Bl L^2
14 y sc^{S1} In49 sc^8; bw; st
15 y sc^{S1} In49 sc^8; sp b cn bw
16 y sc^{S1} In49 sc^8; dp tx I Cy Ins 05 pr cn^2/Bl L^2

London, England: University College, Department of Zoology

Wild Stocks

Kenya (inversion on X)
Oregon

Chromosome 1

BB
Bx^3
N^8/dl-49 y Hw m^2 g^4
ras f
rst^3
v

w
w^{a} m
w^{bl} wy f
w^{e2}
w^{m4}
w sn B
y m car
y and oc pt y pr shf
y and sc rb ct m
y and w^{a} m f
y and y rst^{3} m car

Chromosome 2

bw
cn
cn bw
dp^{v2}
ho
Pt/Cy
vg

Chromosome 3

Mc/Sb
Sb ss^{a}
ss
ss^{a}
st
ve
ve ss^{a}

Multichromosomal

v; bw

Manchester, England: Christie Hospital and Holt Radium Institute Cytogenetics Laboratory

Wild Stocks

Birmingham

Inbred lines (number of generations in brackets)

Oregon v (156)
bpr (66)
dp (57)
e (63)

Chromosome 1

v/y v f car
Muller-5
sc ec cv ct v g f/y Hw m g delta 49
ClB/sc v f car

Chromosome 2

b pr vg
dp b cn bw
Cy/al sp b pr c px sp
dp^{Th} Cy cn bw/S Sp cn bw
dp^{Th} Cy, In-L pr cn^{2} In-Cy R-O/Ins-NSL Ins-NSR px sp (Cy Oster)

Chromosome 3

Mc/Sb

Multiple

dp; e
CyL/Pm; H/Sb

Sheffield, England: The University, Department of Genetics

Wild Stocks

Bacup
Bannerdale
Bayfordbury-2
Dronfield
Oregon (Davis)
Oregon-Samarkand

Inbred

Bayforbury-1
CyL/Pm
Isocanton
Oregon-1
Oregon-2
y bw st v

Chromosome 1

B
br
ClB/sc v f car
ct^{6} v bi
f^{55f}
g
Muller-5
v
w
w m B
w y
w^{a}
w^{a} v
w^{a} and =: y
y

Chromosome 2

bw
bw cn
cn
Cy L/Pm (inbred)
Cy/L^{2}
po vg
stw^{3}
vg
vg^{no}
vg^{p}
Ly/LVM
p^{53h}
p^{p}
se
se cp e
st
th st cp
ve h th

Chromosome 3

e
e^{11}
D/LVM
Gl Sb/LVM

Chromosome 4

ey^{R}

Multiples

bw st
v bw
v st
vg bx
y bw st

INDIA

Calcutta: Indian Statistical Institute

Wild Stock

1 Canton-S

Chromosome 1

2 B
3 Bx^{3}
4 cv
5 $Df(1)N^{8}$/dl-49 y Hw m^{2} g^{4}
6 ec
7 f
8 In(1)Cl $sc^{?}$ $t^{?}$ v^{+} $sl^{?}$ B^{36d}/dl-49 y Hw v m^{2} f
9 m
10 v

11 w
12 w^{a}
13 w^{bf}
14 w^{bl}
15 w^{co}
16 w^{e}
17 w^{h}
18 w^{i}
19 y
20 y^{2}

Chromosome 2

21 bw
22 cn
23 dp
24 ho
25 px
26 vg
27 vg bw

Chromosome 3

28 cu
29 e^{s}
30 Ly/D^{3}
31 ry
32 se
33 ss^{a}

Chromosome 4

34 ci^{W}
35 ey^{2}

Multichromosomal

36 Cy/Pm D/Sb

ISRAEL

Jerusalem: Hebrew University of Jerusalem

60 common laboratory stocks
25 second-chromosome lethals balanced over Cy/L
Several wild strains from various parts of Israel
Several isogenic strains derived from wild populations in Israel

ITALY

Milano: Universita' di Milano, Istituto di Genetica

Wild Stocks

1 Canton-S
2 Chieti-v
3 Crkwenika
4 Gaiano
5 Jaslo o. c.
6 Moltrasio
7 Oregon-R
8 Pavia
9 S. Maria
10 Suna
11 Urbana
12 Valdagno
13 Varese

Chromosome 1

14 B
15 fa^{n}
16 N^{B}-S
17 ptg
18 sd
19 w
20 w^{a}
21 w^{bl}
22 w^{e}
23 y Hw

Chromosome 2

24 a px sp
25 ab
26 ast^{4} dp cl
27 b cn vg
28 blt
29 blt^{S}
30 bw ba
31 c wt px
32 cn
33 ft
34 ll^{2}
35 net
36 so
37 so^{2} b cn
38 So^{C}
39 spt

Chromosome 3

40 cp
41 gl^{3}
42 mwh
43 obt
44 ri-s-se ss k e^{s} ro
45 ru b st p^{p} ss e^{s}
46 ru
47 ve

Multichromosomal

48 px^{43j} oo; ru jv se st ca

Not localized

49 tg (formerly abab^{49})

Inversions

Chromosome 1

50 ClB/+
51 ClB y/y Hw m^{2} g^{4}
52 l(1)7/dl49 y Hw m^{2} g^{4}
53 Muller-5

Chromosome 2

54 Cy sp/Pm
55 Cy E-S/S
56 Cy pr/d b
57 M(2)l/In(2R)Cy
58 M(2)B/In(2L)t, l(2)B
59 spd gt-4/Cla

Chromosome 3

60 H/Sb sr In(3R)Mé
61 ltr/Sb sr In(3R)Mé
62 R Ly/In(3R)P, gm

Multichromosomal

63 L^{4} Cy sp/Pm; H/Sb sr In(3R)Mé

Deficiencies

64 Df(2)Px^{2}	Df(2)Px^{2}, bw sp/SM1, al^{2} Cy sp^{2}
65 Df(2)bw^{5}	Df(2)bw^{5} sp^{2}/Xa
66 Df(2)Px	Df(2)Px/Df(2)P; Dp(2;3)P/In (3R)Mo, sr; w^{e}

Stocks selected for tumor manifestation

67 tu A1
68 tu B1
69 tu B2
70 tu B3
71 tu B4
72 tu C1
73 tu C2
74 tu C3
75 tu C4
76 tu C5
77 tu D
78 tu Aspra
79 tu mwh
80 tu Oregon
81 tu SoC
82 tu w
83 tu y Hw
84 e 144 melanotic

Naples: University of Naples, Institute of Genetics

Wild Stocks

Bisignano
Canton
Lecce
Oregon-R
Pavia
Perugia
Roma
Sciolze

Chromosome 1

B
v
w
w^{a}
w^{bf}
w^{bl}
w^{co}
w^{i}
w^{t}
ClB/w^{bl}

Chromosome 2

b cn vg
bw
cn
Cy/Pm
vg

Chromosome 3

cp
Dfd/Cx, D
Gl
Mc
Sb
se
ss^{a}
se ri ss k e^{s} ro
H

Chromosome 4

sv

Multichromosomal

px^{43j} oo; ru jv se
st ca

Special Stocks

Oregon (from 380 generations brother-sister matings)
A stock with a strong Pod effect

Unanalyzed

A modifier of vg
Pm/l
undulated wings

Pavia: University of Pavia, Institute of Genetics

Wild Stocks

1 Ankara
2 Canton-S
3 Chieti
4 Crkvenica
5 Jaslo
6 Oregon-R
7 Pavia
8 Suna

Chromosome 1

9 B
10 Df(1)N^{8}/In(1)dl49, y Hw m^{2} g^{4}
11 ec^{60e}
12 f^{36a}
13 Ins(1)sc^{S1L}, S, sc^{8R}, w^{a} B (=Muller-5)
14 In(1)w^{m4}
15 lz/ClB
16 sc ct v f car/ClB
17 t
18 w
19 w B
20 w^{a}
21 w^{bf}
22 w^{co} sn
23 w^{e}
24 w^{i}
25 w^{i} f^{3} bb^{N}
26 w^{t}
27 y w^{a} cv v f
28 y^{1}
29 y w sn

Chromosome Y

30 f.Y^{S}/sc Y^{L} & y w f/sc Y^{L}

Chromosome 2

31 b cn vg
32 b el rd^{S} pr cn
33 b lt wxt bw
34 Bl L/Cy
35 L^{2}/Cy
36 ltd
37 nub^{2}
38 oo
39 sf^{2}
40 shv
41 so
42 vi

Chromosome 3

43 bx^{34e}
44 cd
45 cd^{45j}
46 D^{3} Sb ca^{3}/Payne
47 Dfd/In(3LR)Cx, D
48 Dl^{7}/In(3R)Mé Sb sr
49 e^{11}
50 e^{11} cd^{45j}
51 fj-s
52 Gl Sb/LVM
53 in
54 ri ss k e^{s} cd^{45j}
55 rs^{2} h^{2}
56 rsd
57 se
58 se h^{1} app
59 se ri ss k e^{s}
60 ss
61 ss^{a44a}
62 st
63 st ss e^{4}
64 tt wo
65 tx^{52j}
66 Ubx, e^{4}/Payne, Dfd ca
67 ve h th
68 wo

Chromosome 4

69 ey^{2}
70 pol
71 sv

Multichromosomal

72 al L^{4} Cy sp/Pm; H/Sb sr In(3R)Mé (2;3)
73 bw; e^{4} wo ro (2;3)
74 cn; ss (2;3)
75 px^{43j}; jv se ca (2;3)
76 sf^{2}; ss (2;3)
77 shv; cd^{45j} (2;3)

78 shv; e^{11} (2;3)
79 shv; ss (2;3)
80 ClB/+; al L^4 Cy sp/Pm; H/Sb sr In(3R)Me (1;2;3)
81 lt stw^3; ri; ey^2 (2;3;4)
82 pk; sp; ey^2 (2;3;4)
83 SM1, al Cy sp^2/Sp; H/Ubx^{130} e^s; pol (2;3;4)
84 l(1)55aB/FM4, y^{31d} sc^8 dm B; SM1, Cy/Sp; Sb/Ubx; pol (1;2;3;4)
85 sc^{S1} B InS w^a/sc^8; SM1, al Cy sp^2/dp b Pm ds^{33k}; C Sb/Ubx^{130} e^s; pol (1;2;3;4)

Translocations

86 ss bxd k e^s/Xa (2;3)
87 Cy SM1; Ubx^{130}/Xa (2;3)
88 pr cn/T(Y;2)C (Y;2)

Attached-X

89 FMA3, y^2/FM6, y^{31d} sc^8 dm B
90 y/+
91 y f:=/X^c, ec f

Closed-X

* X^c, ec f ....(91)
92 X^{c2}, cv v f
93 X^{c2}, y^{49}

Triploids

94 y^2 sc w^a ec/FM6, y^{31d}, sc^8 dm B

Note: For description of Attached-2R;2L and Attached-3R;3L see this DIS, New Mutants report of I. E. Rasmussen.

Attached-2R;2L

95 2R, cn bw;2L, b

Attached-3R;3L

96 3R;3L, rs^2 h^2

JAPAN

Anzyo, Aichi: Nagoya University, Faculty of Agriculture

Wild Stocks

1 Anzyo-Aichi
2 Canton Special
3 Chausuyama-Aichi
4 Hachijojima
5 Hikosan-Kyushu
6 Hiroshima
7 Hita-Kyushu
8 Omogo-Shikoku
9 Oregon
10 Shioya-Hokkaido
11 Suzuka-Mie
12 Takagicho-Tokyo
13 Yonekawa-Yamaguchi

Chromosome 1

14 Bx
15 ec ct^6 g^2 bb^1/ClB
16 f
17 m
18 m^{58i}
19 sc^{S1} B InS w^a sc^8 (Muller-5)
20 sc^{S1} B InS w^a sc^8 $l^{(1)59}$/y w m f
21 v
22 w
23 y^{521}
24 y w m
25 y w m f
26 y w m f/y ClB

Chromosome 2

27 b
28 bw
29 bw (from population bell No. 33)
30 cn
31 Cy/bw (M)
32 Cy/bw (T)
33 Cy bw/bw
34 Cy/$l^{(2)50c}$
35 Cy bw/$l^{(2)50c}$
36 Cy/Pm
37 dp^x
38 dp^v b
39 Pm b
40 Pm/$l^{(2)50c}$
41 vg
42 vg^{Nw} Hia/T(2;3)

Chromosome 3

43 cu
44 e
45 Sb

Multichromosomel

46 Cy/$l^{(2)50c}$; Sb
47 Cy/$l^{(2)50c}$; Sb cu/cu
48 Pm/$l^{(2)50c}$; cu
49 v; bw

Unanalyzed

50 Minute-like
51 Dichaete-like
52 brown-like
53 jaunty-like

Chiba-Shi: National Institute of Radiological Sciences, Biology Division

Wild Stocks

001 Bikini-Atoll
002 Canton-S
003 Idaho-Falls
004 Misima
005 Miyazu
006 OR347(iso)
007 Orinda-I
008 Riverside
009 Samarkand
010 Samarkand
011 DK429(iso)
012 Swedish-b-6
013 Urbana-S'
013 Itabashi
014 Kurosuna
015 Anagawa
016 Hanazono
017 Masutomi

018 Nagasaki
019 Hakata-1
020 Hakata-2
021 Kagoshima

Chromosome 1

101 B
102 ec ct^{6} g^{2} bb^{1}/ClB

103 g^{2} ty/y
104 Muller-5
105 v
106 w
107 y w f m

Chromosome 1

201 b pr Bl
202 bw
203 cn
204 cn wt
205 dp
206 S/Cy E-S
207 vg

Chromosome 3

301 cu kar
302 e^{11}
303 se
304 se ry

Chromosome 4

401 ci^{D}/Cat

Multichromosomal

501 Muller-5; Cy/Pm; Sb/Ubx
503 y; bw; e; ci ey^{R}
504 st; bw
505 cn; ca; gvl
506 X^{c2}, v & y:= (bw; e; ey)
507 y sc^{S1} In49 sc^{8}; bw; st p^{p} (from Prof. Muller)
508 sc^{8}.Y/y In49 B ♂ & y f:= ♀ ; bw^{D} (from Prof. Muller)
509 sc^{8}.Y/y B ♂ & y f:= ♀ (from Prof. Muller)

Kyoto: Kyoto University, Faculty of Science, Department of Zoology

Wild Stocks

Common stocks

1 Canton-S
2 Oregon-RS

From different natural populations

Formosa:

3 Taichu

France:

4 Commes
5 Frasne
6 Forgeneuve
7 Gif
8 Montalivet

Israel:

9 Eilon
10 Giv'at Brenner
11 Qiryat-Anavim

Japan:

12 Anzyo
13 Ashitakayama
14 Ehime-Ishizuchi
15 Ehime-Tambara
16 Erabu-I
17 Erabu-II
18 Fushimi
19 Hirakata
20 Hiroshima
21 Joyama
22 Juriki
23 Kagoshima
24 Kameoka
25 Kyoto-I
26 Marufugi
27 Minoo
28 Miyakonojo
29 Miyazu
30 Naze
31 Saikyo
32 Sapporo
33 Shirahama
34 Shodoshima
35 Suyama-I
36 Tokushima
37 Tokyo
38 Wakayama
39 Yamaguchi
40 Yonago

South Africa:

41 Bloemfontain
42 Tzaneen
43 Umgazi River-I
44 Umgazi River-II
45 Umgazi River-III
46 Western C. P.

Switzerland:

47 Bulligues

Chromosome 1

48 B
49 ec ct^{6} g^{2} bb^{1}/ClB
50 f
51 sc^{S1} B InS w^{a} sc^{8} (Muller-5)
52 v
53 w
54 w^{a}
55 w^{e}
56 w m
57 y
58 y w f
59 y w m f

Chromosome 2

60 b
61 b gp
62 b vg
63 bw
64 bw (Nanzenji)
65 bw^{4}
66 cn
67 cn bw
68 dp
69 dp^{x}
70 L^{2}
71 S/Cy E-S
72 vg
73 vg^{no}
74 wt

Chromosome 3

75 bar-3
76 e^{11}
77 e^{11} Hn^{r3}
78 e^{11} se
79 se
80 se (Nanzenji)
81 Hn^{r3}
82 ss^{a}
83 st
84 wo

Chromosome 4

85 ci ey^{R}
86 sv^{n}

Multichromosomal

1;2

87 v; bw
88 v; dp

1;3

89 w; e^{11}
90 v; bar-3

2;3

91 bw; e
92 bw; st
93 dp; bar-3
94 vg; bar-3
95 vg; Hn^{r3}

2;3;4

96 cn; ca; gvl

1;2;3;4

97 y; bw; e; ci ey^{R}

Lethal Stocks

98 l-(1)TS-45(X-ray induced)
99 l-(1)TS-56(" ")
100 l-(1)57-3014(")
101 l-(1)57-3034(")
102 l-(1)us-2(ultra sonic induced)
103 l-(1)us-6(" ")
104 l-(1)us-7(" ")
105 l-(1)us-11(" ")
106 l-(1)us-14(" ")
107 l-(1)us-16(" ")
108 l-(1)us-20(" ")
109 l-(1)us-25(" ")
110 l-(1)us-33(" ")
111 l-(1)us-47(" ")
112 l-(1)us-48(ultra sonic induced)
113 N^{268-8} N^{264-8}/dl-49 y Hw m^2 g^4
114 $N^{264-112}$ (In)y $N^{264-112}$/dl-49 y Hw m^2 g^4
115 w^{258-14} (Df w)y w^{258-14}/dl-49 y Hw m^2 g^4
116 Df(2)px^2/Cy L^4 sp^2

Special Stocks

117 st sr cr ro ca tu^{36e}
118 tu^{36e}
119 tu bw
120 tu st
121 (Y/Basc)/(Y/Miyazu-X/Basc)
122 (Y/w m)/(Y/Miyazu-X/w m) XXY stocks
123 T(2;3)

Misima, Sizuoka-ken: National Institute of Genetics

Wild Stocks

Mass cultures:

1 Anjio
2 Aosima
3 Asitakayama
4 Bayfordbury
5 Bochu
6 Canton-S
7 Hachijozima
8 Hayahosi
9 Hikone
10 Hikosan
11 Hirakata
12 Hiroshima
13 Igurosima
14 Isima
15 Isizuchi
16 Kamasima
17 Kameoka
18 Kamizawa
19 Kammurizima
20 Katsunuma
21 Kirisima
22 Kochi
23 Minoo
24 Misima
25 Miyazaki
26 Miyazu
27 Nibugawacho
28 Oregon-2
29 Oregon-R #200
30 Oregon-R #264
31 Osyoro
32 Otaru
33 Sagawa
34 Samarkand #297
35 Sapporo
36 Sendai
37 Sioya
38 Sirahama
39 Takadai
40 Tanbara
41 Tatebazima
42 Tesima
43 Tokusima
44 Tokyo
45 Tottori
46 Toyama
47 Uwazima
48 Wakayama
49 Yamaguchi
50 Yata
51-57 African strains

Isogenic strains:

58 Hollande-J-f
59 Hollande-K-f
60 Hollande-G-f
61 H-3-C-f
62 H-3-N-f
63 H-3-O-f
64 Kofu-A
65 Kofu-B
66 Oregon-R, generation 320
67 Osyoro, generation 17
68 Samarkand, generation 432

Chromosome 1

101 B
102 BB
103 Bx
104 car
105 cm
106 ct^6 g^2
107 ec ct^6 g^2 bb^1/ClB
108 f
109 fa
110 f B^iB^i/y f:=
111 g^2
112 Muller-5
113 lz^3/y f:=
114 lz^{37h}
115 lz^{37k}/y f:=
116 lz^{50e}
117 rb
118 t
119 t^3
120 v
121 v (coiso)
122 v f
123 w
124 w (coiso)
125 w B
126 w m
127 w lz^{50e}
128 w^a
129 w^e
130 w^e v
131 w^h
132 w^{sat}
133 X^{c2} v y:=
134 y
135 y ac v
136 y w f
137 y w m f
138 $Y^{Lc}/X \cdot Y^S$
139 Y^{Lc}/y w Y^S & y v f (Muller)

Chromosome 2

201 al dp b pr c px sp/Cy, pr (all)
202 b pr Bl
203 bw
204 bw (coiso)
205 cl
206 cl (coiso)
207 cl bw
208 cn
209 cn (coiso)
210 cn bw
211 cn sca
212 cn wt
213 dp
214 dp^X
215 L^2
216 L^4
217 lt std/SM1, al^2 Cy sp^2
218 ltd
219 ras^2
220 sca bw
221 stw^3

Chromosome 3

301 bar-3
302 ca
303 ca (coiso)
304 ca br
305 cu
306 cu kar
307 dke c
308 Dp/In(3L), Payne, In(3R)C, Sb e l(3)e
309 e^{11}
310 e^{11} (coiso)
311 Hn^r (coiso)
312 Hn^{r3} (coiso)
313 Hn^{r3} e^{11}
314 Hn^{r3} ry (not homozygote)
315 Hn^{r3} sr
316 gl-1
317 mah
318 p^p
319 ry
320 ry (coiso)
321 ry e^{11}
322 ry ca
323 se
324 se (coiso)
325 se ca bv

326 se cu
327 se e^{11}
328 se ry
329 se st
330 st

Chromosome 4

401 Cat/ey gvl
402 ci^{D}/Cat
403 ey
404 pol

Multichromosomal

501 B; e^{11}
502 bw; ca
503 bw; Hn^{r3}
504 bw; Hn^{r}
505 bw; ry
506 bw; st
507 cl; ca bv
508 cl; ry
509 cn; ca; gvl
510 cn; gvl
511 cn; e^{11}
512 cn; ry
513 cn; se
514 cn; ss; gvl
515 cn sca; Hn^{r3}
516 lz/dl-49 m^{2} g^{4};
Cy/Pm
517 lz^{50e}; ry
518 sca bw; se cu
519 sca; ry
520 sca; ss^{a}
521 stw^{3}; gl-1
522 stw^{3}; ry
523 v; bw
524 v; e^{11}
525 v f; cl
526 v f; cn bw
527 v f; cn sca; Hn^{r3}
528 v f; ry
529 v f; se
530 v f; se ry
531 v f; se st
532 v f; sca bw;
se cu
533 vg; se
534 w; bw
535 w; ca
536 w; cl
537 w; cn
538 w; Hn^{r3}
539 w; ry
540 w; se
541 w; px
542 y; cl

543 Ins SM1, al Cy sp^{2}/dp b Pm ds^{33k}; C Sb/Ubx^{130} e^{s}
544 Ins SM1, al Cy sp^{2}/dp b Pm ds^{33k}; Ins TM1 Mė ri/Ubx^{130} e^{s}
545 Ins SM1, al Cy sp^{2}/dp b Pm (ds^{33k}); In(3L)P, In(3R)C, Sb e l(3)e/Ubx^{130} e^{s}
546 S Sp Bl L/Cy cn^{2} sp
547 sc^{V2} B y^{3P}; Ins SM1, al Cy sp^{2}/dp b Pm(ds^{33k}); In(3L)P, In(3R)C, Sb e l(3)e/
Ubx^{130} e^{s}
548 sc^{S1} B InS w^{a} sc^{8}; Ins SM1, al Cy sp^{2}/dp b Pm; C Sb/Ubx^{130} e^{s}; pol
549 548-IV to Ore-R-IV (H-42)
550 545-I to Ore-R_{265}-I (H-55)
551 545-I to Samarkand-367-I (H-65)
552 548-I to Ore-R_{iso}-I (H-200)

Lethal strains (2nd chromosome)

601-627 X-ray induced
628-687 from natural populations
688-724 semilethal from natural populations

Osaka: Osaka University, Faculty of Medicine, Department of Genetics

Wild Stocks

1 Canton-S
2 Oregon
3 Oregon-R (I)

Chromosome 1

101 B
102 B bb
103 car
104 cm
105 f
106 lz^{50e}
107 ras^{2}
108 rb
109 sc^{S1} B InS w^{a}
sc^{8} (Muller-5)
110 sc^{V2} B y^{3P}
111 t
112 t^{3}
113 v
114 w
115 w m
116 w^{a}
117 w^{bf}
118 w^{co} sn^{2}
119 w^{col}
120 w^{e} v
121 y
122 y Oregon-R (I)
123 y w f
124 y w w^{a}
125 y^{S1} sc^{8} B In(1)49

Chromosome 2

201 al b c sp^{2}
202 b gp
203 bw
204 bw^{D}
205 cl
206 cn
207 cn bw
208 cn sca
209 cn vg bw
210 cn wt
211 cn fes(2)K bw/Cy
212 fes(2)adp/al^{2}
Cy sp^{2}
213 ft
214 Gla/Cy, pr
215 L^{2}
216 l(2)me/SM1,
al^{2} Cy sp^{2}
217 px
218 stw^{3}
219 Tft/Cy
220 vg
221 vg bw
222 vg^{U}/Roi bw sp^{2}
or

Chromosome 3

301 bar-3
302 bar-3 bv
303 ca
304 ca bv
305 ca^{2}
306 cd
307 Dl^{Of}/Ubx^{130} e^{s}
308 Dp/In(3L)P,
In(3R)C,
Sb e l(3)e
309 e^{11}
310 Gl Sb/LVM
311 Gl Sb H/Payne
312 gl-1
313 Hn^{r} h ri
314 jv
315 ld
316 N-X/Mė, In(3L)
In(3R)C e l(3)e
317 ro
318 ru h (cu) cd ca
319 ry
320 se
321 se ss
322 se st
323 ss
324 ss bar-3
325 ss^{a}
326 st
327 st ss
328 st ss e^{11}
329 tx^{52j}
330 ve
331 wo

Chromosome 4

401 ci^{D}/Cat
402 ey^{2}

403 ey^{D}/Cat
404 gvl
405 pol

Multichromosomal

1;2

501 v; bw

2;3

502 b; se
503 b; tx
504 bw; ss cd^{57k}
505 bw; st
506 bw; st ss
507 cl^{57j}; ss^{a}
508 cn; bar-3
509 pr; ss
510 stw^{3}; gl-1
511 vg; se
512 Pm, dp b; In(3R)C, Sb e l(3)e/Xa
513 Pm, dp b/Ins(2L+2R)Cy, S^{2}; In(3L)P, In(3R)C, Sb e l(3)e/Ubx^{130} e^{s}
514 Pm, dp b/Ins(2L+2R)Cy, S^{2}; Dl^{0f}/Ubx^{130} e^{s}
515 SM1, al Cy sp^{2}; In(3L)P, In(3R)C, Sb e l(3)e/Xa
516 SM1, al Cy sp^{2}; Ubx^{130} e^{s}/Xa
517 SM1, al Cy sp^{2}/dp b Pm ds^{33k}; In(3L)P, In(3R)C, Sb e l(3)e/Ubx^{130} e^{s}
518 SM1, al Cy sp^{2}/dp b Pm ds^{33k}; TM1, Me ri/Ubx^{130} e^{s}

2;4

519 ft; pol

2;3;4

520 bw; st; sv^{n}
521 cn; bar-3; gvl
522 cn; ss; gvl
523 Ubx^{130} e^{s}/Xa; ey^{D}/Cat

Amylase

601 H-AS (Amy)
602 H-AW (amy)
603 cn Amy bw
604 cn amy bw

Tumors

701 bw; tu
702 tu^{h}
703 v; tu
704 tu; st
705 tu^{36e}
706 tu^{g}
707 st sr es ro ca tu^{36e}

Insecticide-resistant

801 Hikone-R (multiple) (Japan)
802 NS-R (nicotine) (Japan)
803 0.28(2) (HCN) (England)
804 WMB (DDT, parathion) (Japan)
805 WMD (dipterex, DDT) (Japan)
806 WMH (BHC, DDT) (Japan)
807 KSL (parathion, DDT) (Sweden)
808 STSM-1 (U.S.A.)
809 HL2-Top (U.S.A.)
810 HL1-Q (U.S.A.)
811 HL1-Q (U.S.A.)
812 TG-57 (Korea)

Special Stocks

Attached-X

901 yy (Oregon)

Triploids (No selection)

902 bb/+ & + (Philadelphia)
903 3N (Philadelphia)
904 3N wild type (Panama)
905 y^{2}; ru ca (Panama)

Sapporo: Hokkaido University, Department of Zoology

Wild Stocks

Oregon-R
Sapporo (3 strains)
Otaru

Mutants

B, Cy, v, se, w, cn

Tokyo: Tokyo Metropolitan University, Department of Biology

Wild Stocks

1 Aoshima
2 Bochu
3 Canton-S
4 Hachijojima
5 Oregon
6 Oregon-K
7 Oregon-R
8 Sagawa
9 Samarkand
10 Shioya
11 Tokyo

Chromosome 1

12 B
13 BB
14 bb Y^{bb}
15 ec ct^{6} g^{2} bb^{1}/ClB
16 f B^{i}B^{i}/y f:=
17 fw^{501}
18 m
19 sc^{S1} B Ins w^{a} sc^{8} (Muller-5)
20 sd mc
21 v
22 v f B $\frac{\mathrm{XY/y^{2}\ su\text{-}w^{a}}}{\mathrm{w^{a}\ bb}}$
23 w
24 w m
25 w^{a}
26 w^{a} m
27 w^{e}
28 X^{c2} v & y:= (bw; e; ey)

29 y
30 y w m f

Chromosome 2

31 al b pr cn vg a sp/Cy sn^{2} L^{4} sp^{2} (albasp)
32 al dp b pr c px sp/Cy pr (all)
33 ap^{4}/Cy
34 bw
35 bw/Cy
36 cn
37 cn bw
38 conditioned lethal/Cy (Yoshida)
39 Cy
40 dp bw
41 dp^{x}
42 L^{2}
43 L^{5}
44 L^{52c} vg^{no}
45 l(2)^{50}/Cy (Nozawa)
46 S Sp ab^{2} ltd/NS, px ap
47 str 3104
48 vg
49 vg^{ni}
50 vg^{nG}
51 vg^{no}
52 vg^{np}

Chromosome 3

53 Bd^{491}
54 Bxl/Payne, Dfd ca
55 cu
56 Dl^{3}/In(3R)C, E
57 Dl^{491}/l(3)
58 e^{11}
59 Gl^{53b17} H/LVM
60 Sd/l(3) (domin. vg-)
61 se
62 st
63 ve
64 N-X/Xa

Chromosome 4

65 Cat/gvl ey^{R}
66 ci^{D}/Cat
67 Scn/ey^{D}
68 sv^{n}

Multichromosomal

69 b; tx
70 v; bw
71 vg; se

Unanalysed

72 (bx ct)-like
73 intensifier of Bd

KOREA

Kongju, Chung Cheong Nam Do: Kongju National Teachers' College, Department of Biology

Wild Stocks	Chromosome 1	Chromosome 2	Chromosome 3	Inversion
1 Oregon-R	y	cn	se	Muller-5
2 Urbana-S	w	b	e^{11}	
3 Kongju	m	j	Pr	
	B	vg	ro	
	Bx			

Kwangju: National Chunnam University, Department of Biology

Wild Stocks

1 Kongjoo (Korea)
2 Kwangju (Korea)
3 Mokpo (Korea)
4 Najoo (Korea)
5 Oregon-R
6 Quilpart (Korea)
7 Seoul (Korea)
8 Swedish-C
9 Taegoo (Korea)

Chromosome 1

10 BB
11 Bx
12 t
13 v/ClB
14 w
15 y
16 y^{2} cv v f

Chromosome 2

17 al b c sp^{2}
18 bj
19 Bl/Cy, bw^{45a} sp^{2} or^{45a}
20 Bl L^{2}/Cy, bw sp^{2}
21 Bl L^{2}/Cy, sp^{2}
22 c
23 cn
24 ds S G b pr/Cy, L^{2}
25 j
26 Pfd/Ins(2L+2R)Cy, S^{2}
27 Pfd/SM-5
28 Pfd Pin/Cy, sp^{2}
29 Pin
30 pr
31 Sp bw^{D}/SM-5
32 Sp J/SM-5, al^{2} Cy lt^{v} sp^{2}
33 Sp J Pin/Cy, sp^{2}
34 Sp J L^{2}/Cy, sp^{2}
35 S Sp Bl Pfd bw^{D}/Cy, Ins

Chromosome 3

36 ca
37 D/Gl
38 R Ly/Ins(3L)P, gm
39 ro
40 ru
41 ru cu ca
42 ru h th st cu sr e^{s} ca
43 se h
44 Sb H/Ins(3R)C, cd
45 th st cp
46 ve h th
47 W Sb/Ins(3LR)Cx

Chromosome 4

48 ci gvl bt
49 pol

Attached-X

50 y/g^{2} ty
51 Y^{L}/y w Y^{S} & y v f

Inversion

52 Muller-5

Seoul: Seoul National University, Department of Biology

Wild Stocks

Seoul (Korea)
Kang Wha (Korea)
Quilpart (Korea)

Chromosome 1

BB
Bx
w
y
y w
spl
w Bx

Chromosome 2

bj
Bl L^2/Cy
c
cn
j
vg

Chromosome 3

e se
e^{11} se
ru
ro

Inversion

Muller-5

Multichromosomal

y vg

Seoul: Sungkyun-Kwan University, Department of Biology

Wild Stocks

1 Oregon-R
2 Seoul (Korea)
3 Swedish-C

Chromosome 1

4 BB
5 t
6 v/ClB
7 w
8 y
9 y^2 cv v f

Chromosome 2

10 Bl L^2/Cy, sp^2
11 c
12 cn
13 ds S G b pr/Cy, L^2
14 j
15 Pfd/Ins(2L+2R)Cy, S^2
16 Pfd Pin/Cy sp^2
17 Sp J Pin/Cy, sp^2

Chromosome 3

18 ca
19 ro
20 ru
21 ru cu ca
22 ru h th st cu sr e^s ca
23 th st cp
24 ve h th
25 W Sb/Ins(3LR)Cx

Chromosome 4

26 ci gvl bt
27 pol

Attached-X

28 y/g^2 ty

Inversion

29 Muller-5

Seoul: Yonsei University, Department of Biology

Wild Stocks

1 Mokpo (Korea)
2 Najoo (Korea)
3 Oregon-R
4 Seoul (Korea)
5 Swedish-C
6 Taegoo (Korea)

Chromosome 1

7 BB
8 Bx
9 t
10 v/ClB
11 w
12 y
13 y^2 cv v f

Chromosome 2

14 al b c sp^2
15 Bl L^2/Cy, bw sp^2
16 Bl L^2/Cy, sp^2
17 c
18 cn
19 ds S G b pr/Cy, L^2
20 j
21 Pfd/Ins(2L+2R)Cy, S^2
22 Pfd/SM-5
23 Pfd Pin/Cy sp^2
24 Sp J Pin/Cy, sp^2
25 S Sp Bl Pfd bw^D/Cy, Ins

Chromosome 3

26 ca
27 D/Gl
28 R Ly/Ins(3L)P, gm
29 ro
30 ru
31 ru cu ca
32 ru h th st cu sr e^s ca
33 se h
34 Sb H/Ins(3R)C, cd
35 th st cp
36 ve h th
37 W Sb/Ins(3LR)Cx

Chromosome 4

38 ci gvl bt
40 pol

Attached-X

41 y/g^2 ty
42 Y^L/y w Y^S & y v f

Inversion

43 Muller-5

Special Stocks

44 l(2)TG #57j-1 to #15
45 l(2)QI #57i-1 to #11
46 l(2)NJ #57i-1 to #17
47 l(2)MP #59g-1 to #25
48 l(2)TG #59h-1 to #23

NORWAY

Blindern: University of Oslo, Institute of Genetics

Wild Stocks

1 Amherst
2 Canton
3 Florida 26-24
4 Florida 10
5 Formosa, Japan
6 Lausanne-S
7 Oregon
8 Oslo
9 Samarkand (inbred)

10 Swedish-b-6 (Swedish-b cleaned of inversions)
11 Woodbury

Chromosome 1

12 B
13 Bx^{3}
14 ec ct^{6} v g^{3}/ClB
15 f
16 sc cv v f
17 sc w^{Bwx}
18 sc^{S1} B InS w^{a} sc^{8}
19 w
20 w m f
21 w^{a}
22 w^{Bwx}
23 y
24 y ec ct^{6} v f
25 y l(1)7/sc^{S1} B InS w^{a} sc^{8}
26 y pn
27 y w

Altered Y's

28 sc^{8} Y; w
29 sc^{8} Y; y ac sc pn sn
30 y sc^{8} Y; sc^{S1} B InS w^{a} sc^{8}

Chromosome 2

31 al^{2} Cy, InL, lt^{3}/b pr Bl lt^{3} InCyR L^{4} sp^{2}
32 al dp b pr c px sp
33 al dp b pr c px sp/Cy al^{2} lt^{3} L^{4} sp^{2}
34 b j pr cn
35 bw
36 cn bw
37 dp^{51f26}
38 fj px sp
39 j^{49k}
40 j^{50e5}
41 L^{2}/Cy
42 M(2)50j7/Cy, al^{2} lt^{3} L^{4} sp^{2}
43 Pm/Cy, al^{2} lt^{3} L^{4} sp^{2}
44 pr en
45 S^{37b}/Cy
46 vg

Chromosome 3

47 red
48 e^{4} wo ro
49 gl^{3}
50 Gl Sb/LVM
51 jv Hn^{r} h
52 M(3)w/In(3R)C, e l(3)e
53 ma fl
54 p^{42a}
55 ri
56 se
57 st
58 st e^{2}
59 ve

Chromosome 4

60 ar/ey^{D}
61 ci ey^{R}
62 ey^{2}

Multichromosomal

63 sc^{S1} B InS w^{a} sc^{8}; sp; th
64 bw; st
65 cn bw; e^{11}
66 cn bw; ru h st p^{p} ss e^{s}
67 Cy/Pm; H/S Sb

Deficiencies and Translocation

68 Df(1)N^{8}/dl-49, y Hw m^{2} g^{4}
69 Df(2)Px^{2}, bw sp/Cy L
70 T(2;3)Cy

UNITED ARAB REPUBLIC

Assuit, Egypt: University of Assuit, Department of Genetics

Wild Stocks

Egyptian wild populations:

Alexandria
Assuit
Cairo
Fayoum
Heliopolis
Luxor
Mallawy
Nubareya
Sakha
Wadi Natroon

Stocks from other countries:

Lebanon
Uganda
Oregon-K

NEW MUTANTS

Report of Seymour Abrahamson and Eli Siegel

Dp^A: Drop^A 60d28. 3-99.2. Found among offspring of X-rayed females. Apparently allelic to Dp of Krivshenko (DIS 28). Facet number of the heterozygote about 4. Unlike Dp, Dp^A is homozygous viable, and the homozygote usually shows no facets. Conclusive allelism tests between Dp and Dp^A have not yet been performed.

Report of G. E. Brosseau

Sterile Y chromosomes The Y chromosomes listed below carry X-ray induced mutations (or deletions) for male fertility factors in either Y^L (KL$^-$) or Y^S (KS$^-$). The individual fertility factors are designated by lower case symbols, e.g., kl-1, kl-2, etc. Numbering begins at the centromere end of the complex. There are 5 factors in KL and 2 in KS. In most cases, males with a normal X and a sterile Y are completely sterile. The chromosomes behave as normal y$^+$Y's in all other respects. A large number of sterile Y's of various genotypes are available; only those that are most useful in testing for the presence of male fertility factors are listed here.

Y^{L13}: y$^+$ac$^+$ kl-5$^+$ kl-4$^+$ kl-3$^+$ kl-2$^+$ kl-1$^-$·bb$^+$ KS$^+$
Y^{L37}: y$^+$ac$^+$ kl-5$^+$ kl-4$^+$ kl-3$^+$ kl-2$^-$ kl-1$^+$·bb$^+$ KS$^+$
Y^{L11}: y$^+$ac$^+$ kl-5$^+$ kl-4$^+$ kl-3$^-$ kl-2$^+$ kl-1$^+$·bb$^+$ KS$^+$
Y^{L38}: y$^+$ac$^+$ kl-5$^+$ kl-4$^-$ kl-3$^-$ kl-2$^+$ kl-1$^+$·bb$^+$ KS$^+$
Y^{L36}: y$^+$ac$^+$ kl-5$^-$ kl-4$^-$ kl-3$^+$ kl-2$^+$ kl-1$^+$·bb$^+$ KS$^+$
Y^{L3} : y$^+$ac$^+$ kl-5$^-$ kl-4$^+$ kl-3$^+$ kl-2$^+$ kl-1$^+$·bb$^+$ KS$^+$

Y^{S4} : y$^+$ac$^+$ KL$^+$·bb$^+$ ks-1$^-$ ks-2$^+$
Y^{S5} : y$^+$ac$^+$ KL$^+$·bb$^+$ ks-1$^+$ ks-2$^-$

(Y^{L36} and Y^{L3} allow a very low level of fertility with a normal X.)

Report of Elof A. Carlson

Coi: Coiled Carlson, 57g. 2-48.7 ± 0.2 X-ray induced in Ore-R spermatozoa; a dominant, viable, wing mutation phenotypically similar to an extreme Curly-Dichaete combination. As a heterozygote, Coi/+ is not as extreme as the homozygote, resembling instead Cy/+ or jaunty. Viable with jaunty (2-48.7) but shows no interaction effects. Allelism with jaunty is possible. Excellent viability and fertility. RK1.

Report of C. W. Clancy

bw^{53i}: brown Clancy 53i. 2-104.5. Recessive; arose spontaneously in association with another eye color mutant in In(2LR) 40d chromosome. Seems to be slightly darker than bw^1, and combination with vermilion is slightly pinker than v^1; bw^1.

lz^{551}: lozenge Masterson, 551. 1-27.7. Spontaneous; as single male from stock of In(2LR)40d/Cy, sp^2.; belongs to phenotypic class II of Green and Green (1956). Paper chromatography of eyes reveals trace of red eye pigment complex.

lz^{58d}: lozenge Schreckengost, 58d. 1-27.7. Spontaneous, as several males in stock culture of + Canton-Special. Belongs to phenotypic class II of Green and Green (1956). Paper chromatography of eyes shows trace of red eye pigment complex.

pn^{55}: prune Kivett 55. 1-0.8. Spontaneous; as single male in stock culture of In(2LR)40d/Cy, sp^2. A dark prune without mottling or pitting of eye.

y^{59c}: yellow Clance, 59c. 1-0.0. Spontaneous; as single male in stock of In(2LR)40d/Cy, sp; e^{11}. Fits description of original yellow.

Report of S. Counce

fd(2)adp: female sterile(2)adipose Counce, 1956. 2-83±. Pub. Doane, 1959, Genetics 44:506; developmental and physiological studies, Doane, 1960, Ph. D. Thesis, Yale Univ. Spontaneous in Kaduna wild stock maintained in Edinburgh. Adult fat body hypertrophies as cells become distorted by enormous oil globules. Abnormal fat bodies visible through body wall of 6-day old or older adults when submerged in 95% alcohol, then water. Adult corpus allatum of mated females hypertrophies. Females completely sterile, sterility autonomous. Eggs laid by homozygotes show meiotic and/or mitotic abnormalities and never develop beyond early cleavage stages. Males 78% fertile. Heterozygotes fertile, but females become sterile with age. Viability generally good, but longevity reduced; homozygotes with selective advantage under starvation; heterozygotes superior under desiccation. Average water content of well fed adults reduced, while percentage of lipids, as a function of dry body weight, is almost double that of wild type. Iodine numbers show greater degree of saturation of mutant lipid extracts than of wild type. RK2

Report of W. W. Doane

fg: flag Doane, 60f14. 2-20. Unpublished. Recessive; arose spontaneously in Oregon-R wild stock. Wings reduced to 2/3 the length and 3/4 the width of wild type, and held tent-like over abdomen; alulae absent or vestigial; proximal posterior wing margin often irregular with tendency to fold under about longitudinal vein VI; venation usually normal, with occasional blistering. Location still approximate. Good viability and fertility. RK1

Report of Margaret Edmondson

$fs(2)^{58g}$: female-sterile in the second chromosome 58g Edmondson, 1958. 2-35.6±0.5. Ultra-violet induced in b bw stock. The homozygous b bw females are of normal viability but completely sterile. Numerous infertile eggs are produced. Males have normal fertility. No visible effects. Balanced over al^2 Cy Bl cn^2 L^4 sp^2. RK1

Report of O. G. and M. J. Fahmy

elr: elliptical-rough Fahmy, 1956. 1-25.1±0.9. Induced by X-radiation. Eyes slightly elliptical and rough. Wings slightly broader. Males and females viable and fertile. RK1
Alleles: 1 allele induced by X-radiation, 1 allele induced by N-mustard CB. 3025.

sct: scooped-thickvein Fahmy, 1956. 1-16.0±0.7. Induced by methanesulphonate CB. 1540. Short scooped wings, with the inner costal margin frequently incised in several places and veins thickened. Eyes slightly altered in shape and darker. Abdominal tergites slightly ridged. Males sterile, viability about 0.4 wild type. RK2

swb: strawberry Fahmy, 1958. 1-2.2±0.6. Induced by N-mustard CB. 3025. Large rough eye with semi-glazed surface; the irregular pigment distribution gives a patchy brighter red colour. Approximately 2/3 of the flies show varyingly sized incisions of the inner wing margin. Males viable and fertile, female fertility reduced. RK2

unc: unco-ordinated Fahmy, 1954. 1-68.9±1.2. Induced by N-mustard CB. 3025. Wings upheld and frequently partially curled over at tips. Complete lack of co-ordination in movement of legs, most flies being unable to walk and dying shortly after eclosion. RK2

Report of Sara H. Frye

y^{4C}: fourth complementing allele of yellow Origin unknown, present in the sc^{S1} B InS chromosome (b136 in the Bloomington stock list). When y^{4C} is combined with y^2 the heterozygous female is completely normal in phenotype, when with y^{Si} (a spontaneous yellow in the scute-8 chromosome) the phenotype of the heterozygous female is pale yellow with tannish bristles. y^{4C} males have pale yellow wings and body color; their bristles are not as dark as those of y^2 males. Three other alleles of yellow (two of which were X-ray induced) are known to complement with $yellow^2$ (Stone, 1935, Neuhaus, 1936, Sandler, 1960, personal communication).

Report of E. H. Grell

lys: lysine E. H. Grell, 1957. 2-22.9±. Pub. Grell, 1958, Proceedings of the X International Congress of Genetics, Vol. II, 104-105. Spontaneous in stock of rc which was found to be entirely lys rc. When homozygous and by itself lys is a biochemical mutant in which an accumulation of lysine can be detected by chromatography of larvae, pupae or adults. Flies homozygous for lys occasionally have faintly reddish fat cells especially in the thorax. This tendency is enhanced by starvation of the larvae or by combining with the rc or rc^2 mutants or (according to E. B. Lewis, personal communication) the eye color, cho. RK4

rc: red cells Lewis, 1946. 27.3± (relocated from 26+ by E. H. Grell). Formerly known as red blood cells (rbc)(Lewis, DIS 24: 59). Pub. including new name and symbol by Jones and Lewis, 1957, Biol. Bull. 112: 220-4; E. H. Grell, 1958, Proceedings of the X International Congress of Genetics, Vol. II, 104-5. Phenotypic effects described for rc by Jones and Lewis (loc. cit.) result only when flies are homozygous for rc and lys (see this DIS for description of lys). RK5

rc^2: red cells-2 R. F. Grell, 1957. 2-27.3±. Unpublished. Spontaneous in sn^4 stock. Overlaps wild-type at 25° C on standard media; at 17° C a few red fat cells appear in adults. When larvae are starved, many red fat cells appear in the late pupae and adults. Red fat cells are produced when rc^2 is combined with lys. A stronger allele than rc. RK4

Pin^2: Pin-2 E. H. Grell, 57b. 2-107.3±. Spontaneous. The thoracic bristles of the heterozygote are greatly reduced at 25° C. At 17° C the heterozygote appears normal. The homozygote is usually lethal. The rare survivor has almost no thoracic bristles at either temperature. Pin^2/Pin has smaller bristles than Pin^2/+ and has low viability. The locus appears to be within the Px and Px^4 deficiencies as Pin^2/Px and Pin^2/Px^4 are lethal. The Pin$^+$ locus appears to be located in the bx$^+$Y and males of the constitution, bw$^+$Y; Pin^2/+, have thoracic bristles about 1/2 as long as wild type. RK1

Pu^2: Punch-2 E. H. Grell, 57b. 2-97. Spontaneous. Heterozygote has purplish eye color resembling pr. Homozygote is lethal. Pu^2/Pu and Pu^2/T(2;3)p^{Gr} are also lethal. The mutant does not appear to be associated with a chromosomal rearrangement. RK1

Elp: Ellipse E. H. Grell, 57b. 2-99. Spontaneous. Eyes of heterozygotes are rough and more oval than normal. Homozygote has small eyes with fused facets which resemble homozygous ast. Homozygote tends to have low viability. RK1 as heterozygote.

Report of Anita Y. Hessler

lix: little isoxanthopterin Hessler, 1959. 1-23 . (Linkage data of J. L. Hubby.) Originally isolated from a laboratory Oregon-R stock at University of Chicago, Department of Zoology, this mutant is characterized by two factors: (1) extremely small amounts of isoxanthopterin in the male's testis sheath, compared with normal Oregon-R males and (2) accumulation of an unknown substance, possibly a pteridine, in significantly larger amounts than found in testis sheath of normal Oregon-R males. These characteristics of the male testis sheath were recognized chromatographically. Otherwise, these males are undistinguishable, macroscopically and alive, from normal Oregon-R.

Report of W. F. Hollander

rux^{60d}: roughex Rolfes, 1960. Spontaneous. Eyes variably roughened, little reduction in size usually. Both sexes fertile but viability about .5 or less of wild. RK3

(1) A spontaneous mutation from apricot to white eye in Basc, 1960.

(2) A spontaneous mutation to clot? Found 1958 in wild stock collected in Ames. Linkage test only with dp so far, agrees with cl. RK1

(3) A spontaneous mutation to kidney? Found 1957. Preliminary linkage tests indicate probably k. Penetrance and expressivity variable. RK3

Report of E. C. Keller, Jr. and D. J. Nash

A dominant sex-linked club-miniature wing mutant has been obtained from a wild strain of gamma irradiated D.m. Preliminary mapping indicates it is at or near the miniature locus. The mutant is similar to vestigial in the hemizygous male and homozygous female, but like miniature in the heterozygous female. Homozygous females are about 98% non-fecund. Allelic tests with miniature and linkage tests will be continued before assigning a symbol to this mutant.

Report of H. Kikkawa

Amy: Amylase Kikkawa, 1957. 2-80±. Found spontaneously from Hikone wild strain. Strong amylase activities in both digestive and body fluids. Semidominant. (See Research Notes).

fes(2)K: female sterile(2)K Kikkawa, 1960. 2-100±. Found spontaneously from Hikone wild strain. Female sterile. Male entirely fertile. Balanced with Cy stock.

Report of E. B. Lewis

α^{-1}: alpha^{-1} Lewis, 1960. 2-?. Controls dopa oxidase activity. Discovered in a p^p strain. This allele is presumably recessive to two other allelic forms found in Cy/Pm and Samarkand strains. Located 3.6±1.6 units to the left or right of b. Homozygous recessive adults have very little enzymatic activity, modifying genes are not effective and enzyme activity is heat labile relative to wild type enzyme. Good viability and fertility. RK1

TM3: Third Multiple 3 Lewis, 55g. Pub. D. F. Mitchell, 1958 CSH Symp. Quant. Biol. 23:279-290. From repeated X-raying of a Dp(1;3)sc^{260-20} In(3LR)sep, In(3R)C, ri p^p sep bx^{34e} e^s chromosome. A complex balancer for the third chromosome. In addition to the breakage points already present before X-raying, there are new breakage points in six additional regions: 71C, 76C, 79E, 93A, 94D-F and 100C. The new rearrangement appears to be as follows: 1A/61A to 65E/85E to centromere to 79E/100C to 100F/92D to 85E/65E to 71C/94D-F to 93A/76C to 71C/94D-F to 100C/79E to 76C/93A to 92D/100F. The presence of the normal alleles of y and ac serve as terminal markers for the left end of the chromosome and In(3R)C serves as an effectively terminal marker for the right end. Tinderholt (see this DIS) has improved the usefulness of this balancer by inserting the dominant mutants Sb and Ser into it, and has studied its effectiveness in suppressing crossing over. TM3 is associated with a slight Minute bristle effect and has somewhat low viability and fertility.

Report of M. J. Mayo

w^{59j}: white^{59j} X-ray induced in Canton-S sperm. Behaves as an allele of our stock of w. Eye colour nearly snow white. RK1

lz^{58j}: lozenge^{58j} X-ray induced in Canton-S sperm. Eyes smaller than wild type, facets run together. Brown with darker rim. Female sterile. RK1

Report of R. H. McNew

mumac: McNew, 591. Localisation underway. Heat shock induced in wild type male. Males are almost sterile, have wings outstretched, venation somewhat affected. Further tests are being carried out on fertility of both sexes. RK2

Report of Helen U. Meyer

ab^{l-60h}: abrupt-lethal^{60h} Hall, 60h. 2-44.0, locus of abrupt according to Bridges. Our own crossover data, based on 63 crossovers between Sp and b, give the location of the near-lethal allele at 43.0±. Arose spontaneously in a chromosome containing fes ms b cn sp. Rare homozygous survivors and compound ab^2/ab^{l-60h} have shortened vein L5, but no effect on scutellar bristles nor the parted pattern down the midline of the thorax and scutellum. ab^{l-60h}/ab^2 males are fertile, while homozygous ab^2 males are sterile. Normal crossover pattern throughout the entire chromosome. RK3

bur^2: burgundy^2 Hall, 60h. 2-55.3±. Recessive. Arose spontaneously in a chromosome containing fes ms b cn sp. Allele of the UV-induced bur localized at 55.7± by Meyer and Edmondson (DIS-23, 1949). Eye color reddish brown. Brilliant orange color in combination with cn. Normal crossover pattern throughout the entire chromosome. Localization based on 17 crossovers between Bl and cn puts this factor at 55.3±. Good viability. RK1

N^{60c}: Notch-deficiency^{60c} Meyer, 60c. 1-1.5 to 3.0. Affects the loci of w, rst, spl and fa. Arose in X-rayed spermatogonia. Lethal in males. Heterozygous females showed a very good Notch wing effect, but had low fertility. RK3; discarded.

Report of J. D. Mohler

Dp(1;f)60g: Duplication(1;f)60g Mohler, 60g. Spontaneous in triploid female as complement to $\overline{XX}$, Df(1)60g described below.

Fo: Forkoid Mohler, 58c18. 2-107-. X-ray induced. Heterozygotes show reduction in size of bristles and weak forking of head and posterior thoracic bristles. Lethal homozygous. Not included within Df(2)Px. The phenotypic effect is partially suppressed by Dp(2;3)P which presumably carries an active wild type allele. Homozygotes with Dp(2;3)P frequently survive, are sterile and have very extreme forking of all bristles. Fo interacts with alleles of the f locus to produce more extreme forking with the order of expression being +/+; Fo/+<+/f; Fo/+≤f/f; +/+<f/f; Fo/+ =f^{36a}/f^{36a}. Males with f and Fo resemble f^{36a} in phenotype.

$\overline{XX}$, Df(1)60g: Attached-X, Deficiency(1)60g Mohler, 60g. Spontaneous as a single triploid female carrying both XX, Df(1)60g and Dp(1)60g from a free-X triploid line carrying In(1)sc^8, In(1)dl49, y^2 B f v m w^a sc^8 heterozygous. The attached-X is a reversed acrocentric having the proximal X from the sc^8 inversion. The deficiency is viable in 3n females, but is lethal in 2n females and intersexes ($\overline{XX}$Y) in the absence of the complementary duplication. Besides y^2 ac$^+$ from the distal end of the sc^8 chromosome the duplication carries su-f$^+$ (but not car$^+$) which could have come from either chromosome (or perhaps both?).

Report of B. Nicoletti

w^{cf}: white-coffee Nicoletti, 1960. 1-1.5. The mutant white-coffee arose in y^2 ma-l chromosome which had been X irradiated with 4000 r. It was recovered as a brown-eye mutant in a female heterozygous for w^a. Subsequent tests have shown that w^{cf} is a member of the white series. Larval malpighian tubules are yellow. Females heterozygous for w^{cf} and w, w^a, w^{co}, w^{cp}, w^{bl}, w^{col} or w^{sat} are phenotypically w^{cf}. When heterozygous for the wild-type allele, w^{cf} appears to be completely recessive, and in females heterozygous for w^{cf} and homozygous for st, no evidence for a slightly dominant effect of w^{cf} can be found; it therefore differs from w^{Bwx} in this respect (Mossige, J., DIS-27: 59). The w^{cf} eye color, which is the same in males and females, resembles w^{sat} at eclosion but darkens significantly with age so that after a few days post eclosion the eye color has darkened to such an extent that it looks like sepia. Phenotypically this is the darkest allele of the white series. Preliminary paper-chromatographic analysis has shown that w^{cf} has more red pigment than w^{sat}, while w^{Bwx} has little or none. Therefore in the seriation of amount of red pigment found in alleles of white (Morita, T., and T. Tokuyama, DIS-33: 148), w^{cf} would occupy the extreme position after w^{sat}.

A salivary chromosome analysis has not shown any gross rearrangement in the X chromosome. In the following crosses a reduction in recombination was detected between y-w^{cf} as compared to y-w^{a}:$y+/+w^{a}$ = 1.1(26/2368); $y+/+w^{cf}$ = 0.64(16/2493). In an earlier test using y^{2}, the recombination value obtained from $y^{2}w^{cf}/++$ heterozygotes was found to be 0.53(14/2652). Preliminary experiments indicate that pseudoallelic recombination occurs between w^{cf} and w. RK1

Report of B. Nicoletti and E. B. Lewis

Tp(3)Vno: Transposition (3) Vein off New symbol and name to replace In(3R)Vno. Previous salivary gland chromosome analysis by Nicoletti (see report of E. H. Grell DIS 33:94) indicated that the Vein off mutant of Grell was associated with an inversion extending from 89E to 97A. Further analysis confirms these breakage points but indicates an additional breakage point in the region of 93F, probably just preceding 94A1-2. The new rearrangement is a complex transposition having the following sequence: 3L normal through centromere to 89E/93F to 97A/89E to 93F/97A to tip of 3R. The symbol In(3R)Vno should therefore be discarded in favor of Tp(3)Vno. The conclusion still stands that this rearrangement should be effective in reducing pairing in the distal part of the right arm of the third chromosome (see R. F. Grell and E. H. Grell, 1960, Proc. Natl. Acad. Sci., Wash. 46:51-57).

Report of I. E. Rasmussen

Attached-2R;2L Rasmussen 59k.60c. Only a brief description of the stocks, in order to make them available to other workers, will be given here. A detailed report on the synthesis will be given elsewhere. The rearrangements were X-ray induced, independently for 2R and 2L. Breaks are in heterochromatin close to the centromere. The new sequence is as follows: tip of 2R to centromere and section of 2L het./base 2R to tip of 2R; tip of 2L to centromere and section of 2R het./base 2L to tip of 2L. For analogy with attached X's these rearrangements are abbreviated as 2R;2L. They have been synthesized mainly in order to be able to make use of a partial tetrad analysis in the study of certain loci on the major autosomes. New markers may be introduced or over triploids or, very efficiently, by X-raying females of the desired genotypic constitution and crossing them to males of the 2R;2L constitution. The proof that the proper rearrangement has been obtained can obviously only be a cytological one by examining metaphase plates. Attached-3R;3L. 3R by Orias and Deal 58l. 3L Rasmussen 59f. See description of 2R;2L.

ec^{60c}: echinus Lorenzoni 60c. Arose spontaneously in Wild Pavia. Less extreme than ec. Eyes not uniformly rough.

Report of O. S. Reddi

bw^{CB} allele of bw induced in spermatogonia of Drosophila by CB.1506 (2-chloroethyl methane sulphonate). bw^{CB}/bw^{CB}, lethal; bw^{CB}/bw, indistinguishable from bw/bw; bw^{CB}/bw; st/st, produces peach coloured eyes. A cytological change involving more than one or two bands was excluded (Slizynska).

Report of A. Schalet

Ubx^{A}: Ultrabithorax Schalet, 1959. From X-rayed cu kar ♂. Heterozygote shows halteres like Ubx. In addition most flies have a roughening of the eyes which is variable in extent and expression. Most flies lack one or both of the postvertical bristles. A few flies may show a slight upward curvature of the wings. Ubx^{A}/Ubx^{A} and Ubx^{A}/Ubx are lethal. Viability and fertility of heterozygote is good. Ubx^{A} is apparently associated with an inversion (see linkage data this issue), but no cytological examination has been made.

Report of V. Tinderholt

TM3, Ser: Third Multiple 3, Serrate A complex third chromosome balancer obtained by introducing the dominant gene Serrate into TM3 (see Lewis, this DIS). Serrate was introduced, undoubtedly, by double crossing-over when the inversions FM6 and

SM5 were present as the X and second chromosome balancers respectively to increase the frequency of double crossovers. The balancer still carries the recessive genes ri p^p sep bx^{34e} e^s. Penetrance of the Serrate gene is virtually 100% at 25°, but may overlap wild type occasionally at 28°.

The balancer with Serrate effectively suppresses crossing-over throughout the third chromosome when no other inversions are present. When other inversions are present in the background with TM3, crossing-over occurs in the left tip of TM3. When inversions are present in all of the major chromosomes (i.e., the combination FM6, SM5, TM3), crossing-over in the left increases to approximately 18% in the extreme tip of TM3 and drops to 0% twenty crossover units from the tip. Crossing-over is effectively suppressed throughout the rest of the chromosome, even when other inversions are present; only double crossovers may occur and this is less than 0.2% in certain regions of the chromosome. Special care should be observed when TM3 Ser is used with other inversions. Serrate separated from TM3 approximately 1 in 2000 gametes, when FM6;SM5 were present. This event is detectable since e^s is mildly semidominant and the phenotype of TM3 Ser has a slightly dusky body color, with serrated wings, while Serrate alone does not have this phenotype. It is recommended that TM3 Sb Ser be used if complete certainty is desired. Viability and classifiability are very good.

TM3 Sb Ser: Third Multiple 3, Stubble Serrate This is identical with the above except that the dominant gene Stubble has been inserted into TM3 Ser presumably through a rare double crossover. This chromosome has not been tested thoroughly but carries the genes ri, p^p, sep, and e^s. Presence of bx^{34e} has not been tested and is probably absent. Viability is very good and classifiability is excellent.

Report of W. J. Welshons

In(1)pdf: pod-foot Welshons. 57h6. Arose as an w^a pdf male from an irradiated (4000 r) w^a male mated to y w f; Dp w^{51b7}/+ females. Terminal tarsus swollen on one or more legs. Females heterozygous for pdf are wild type. Good viability and fertility in homozygous and hemizygous condition. Rarely overlaps the wild type. Preliminary cytological observations indicate the presence of an X chromosome inversion extending from the region of 16B7 to 19F-20A. RK2A

LINKAGE DATA

Report of E. and J. Krivshenko

Sco: Scutoid (See DIS 33:96). Crossing over data on this second chromosome mutant are now available. In one cross Sco was found to be 1.3 units to the right of black (b) and 3.1 units to the left of purple (pr). A second cross showed Sco as 1.8 units to the right of b and 14.8 to the left of vg.

Combined results give Sco as 1.7 units to the right of b, corresponding to a map position of 51.2 on the second chromosome.

Ps: Pigmentless (See DIS 33:95). This gene appears to be inseparable from cinnabar which arises simultaneously with Ps. Preliminary data on crossing over with other genes shows Ps to be located in the region of cn.

No macro-aberrations are connected with the mutation Ps.

Report of A. Schalet

Relocation of the mutant rosy The mutant ry (rosy) was reported by Bridges (DIS 9, 1938) to be on the third chromosome at 51± between cu (curled) and kar (karmoisin). However, crossovers between kar and ry yield wild-type and cu kar ry Sb flies in the following cross: cu kar + +/+ + ry^2 Sb ♀ X cu kar ry^8 ♂. 0=895+897; 1=23+17; 2=4+3; 3=69+80; N=1988; R1=2.0; R2=0.35; R3=7.5. Consequently, ry may more accurately be placed at about 52.4 just to the right of kar. (The ry^8 allele of the ♂ parents is one of many new rosy mutants induced by X-rays in a cu kar chromosome.)

Relocation of M(3)S34 M34 is listed in Bridges and Brehme as a long-bristled somewhat variable Minute located on the third chromosome at 49±, to the left of cu. A stock carrying this mutant, M(3)S34/T(2;3)Me, was obtained from Pasadena. Although the bristles appeared normal to my eyes, there was a lethal located between Ly (Lyra) and Dfd (Deformed). A more precise localization was obtained from the following cross: M34 ry^2 Sb/Dfd ry^2 Ubx e^4 ♀ X M34 Dfd/Xa ♂. There were 29 Xa$^+$ individuals out of a total of 1847 offspring. Of the 29, 26 showed Sb and 3 showed Ubx. Therefore, the recombination frequency between M34 and Dfd is 29 X 2/1847 or 0.031; and M34 is placed to the left of Dfd at about 44.4. (Parental ♂♂ from the above mating were subsequently crossed to other ♀♀ with no third chromosome markers and this mating produced Xa and Xa$^+$ offspring in approximately equal numbers.)

Relocation of Notch-Xasta The mutant N-X (Notch-Xasta) was reported by E. Ohnishi (DIS 25, 1951) to be on the third chromosome at 50.9. From a mating of N-X Dfd ry^2/cu kar ♀ X cu kar ry^7 ♂, about 13 N-X cu kar appearing offspring appeared among 4,815 counted. There was one fly that appeared to be N-X cu ry. (Under the conditions of this cross, the three mutants N-X, Dfd, and cu overlapped wild-type.) From a mating of M34 Dfd/N-X ry^2 Sb ♀ X cu kar Ubx^A/Xa ♂, in which the Ubx^A offspring were carefully examined and the Xa offspring casually examined, there appeared one N-X Dfd ♀ among 367 Ubx^A flies counted. However, one N-X Dfd ♂ showed up among the uncounted Xa offspring. (The N-X/Xa phenotype is somewhat difficult to distinguish from Xa alone.) Breeding of this ♂ confirmed the phenotypic designation. In a final cross of st/N-X Dfd ♀ X st ♂, the recovery of st N-X Dfd offspring places N-X between st (44.0) and Dfd (47.5).

Crossing over in a third chromosome carrying Ubx^A

ru h st p^P ss e^s/cu kar Ubx^A ♀ X ru h st p^P ss e^s ♂

ru h st p^P ss e^s	405
Ubx^A	513
h st p^P ss e^s	20
ru Ubx^A (not scored)	
st p^P ss e^s	2
ru? h Ubx^A	2
Ubx^A e^s	1
	943

No ru Ubx^A phenotypes could be scored because the Ubx^A alone causes a roughening of the eyes. The Ubx^A chromosome apparently carries an inversion with one break between h and st, but closer to h, and another break to the left of e^s.

STOCK LISTS

AMES, IOWA: IOWA STATE UNIVERSITY
Department of Genetics

D. simulans v
D. simulans y w

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

D. affinis: Amherst 1954
D. busckii: Amherst 1958
D. algonquin: Amherst 1958
D. funebris: Amherst 1954
D. hydei: Amherst 1956
D. immigrans: Amherst 1956
D. melanica: Amherst 1952
D. robusta: Amherst 1960
D. simulans: Wallingford, Pa. 1958

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

D. simulans: wild

D. virilis: Pasadena wild
scarlet

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Zoology

D. americana americana

1 Independence, Ohio (wild)
2 cn
3 o

D. texana

1 New Orleans

D. virilis

Wild Stocks

1 Pasadena
2 Texmelucan, Mexico

Chromosome 1

3 ap^{40e}
4 v^{48b}
5 w^{50112}
6 y^{40a} dy
7 y^{40a} ec ch^2 dy

Chromosome 2

8 va

Chromosome 3

9 gp^2
10 S/+
11 sv
12 sv t tb gp^2
13 sv tb gp^2
14 t
15 tb
16 tb gp^2

Chromosome 4

17 cd

Chromosome 5

18 B^4 pe^{Jap}
19 es B^3
20 es pe
21 pe
22 ru
23 ru st B^3 pe^{Jap}
24 ru st es mh B^3
25 ru st mh
26 ru st mh B^3
27 st B^3
28 st es pe^{Jap}
29 st mh
30 st pe
31 st pe^{Jap}

Multichromosomal

32 b; pe^{Jap}
33 b; tb gp^2; cd; pe
34 cd; es
35 cd; es pe
36 cd; pe
37 cn; es pe
38 cn; pe
39 gp^2; B^3 pe
40 pe; gl
41 t; cd
42 v^{48a}; pe
43 v^{48a} w; pe
44 w; cd; pe
45 w; cn; pe
46 w; pe
47 w; st pe
48 w^{53e}; pe

Peach Mottled Translocations (homozygous)

49 $T(2;5)pe^{m39}$
50 $T(3;5)pe^{m4}$
51 $T(3;5)pe^{m21}$
52 $T(3;5)pe^{m51}$
53 $T(3;5)pe^{m51}$ R(pe) #2; t tb gp^2
54 $T(3;5)pe^{m51}$ R(ru st B^3 pe^+ sv) #2
55 $T(3;5)pe^{m3}$

COLD SPRING HARBOR, NEW YORK: CARNEGIE INSTITUTION OF WASHINGTON

D. busckii: wild-type B
D. pseudoobscura: wild-type, Pinon Chiricahua
D. simulans: Wild-type, South Africa
D. virilis: China-a
D. willistoni: wild-type, Belém

DAYTON, OHIO: UNIVERSITY OF DAYTON

D. persimilis

Wild Stocks

Yosemite National Park, él. 8,000'
Whitney (3 strains)
Klamath (4 ")
Mendocino (4 ")

Chromosome 2

Dl cd

Chromosomes 3 & 4

or Cy

D. robusta

Wild strain from Dayton, Ohio

DeKALB, ILLINOIS: NORTHERN ILLINOIS UNIVERSITY
Department of Biological Sciences

D. robusta: DeKalb wild type
Standard
S136-4S (Steelville, Mo.)

D. virilis: multiple mutant

LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY

D. ananassae: Baracoa, Cuba (1 strain), 1952
D. affinis (strains taken at weekly intervals from June 1960, through September 1960)
D. busckii (1 strain), 1960
D. nigromelanica (2 strains), 1960
D. putrida (strains taken at weekly intervals from May 1960, through September 1960)
D. robusta (strains taken at weekly intervals from May 1960, through September 1960)
D. tripunctata (strains taken at weekly intervals from August 1960, through October 1960)

LINCOLN, NEBRASKA: THE UNIVERSITY OF NEBRASKA
Department of Zoology

D. affinis: Alabama, Georgia, Iowa, Kentucky, Minnesota, Missouri, Nebraska, New York, North Carolina, South Carolina, Tennessee, and Texas. All wild-type strains except for several containing CO_2 sensitivity.
D. algonquin: Minnesota and Nebraska
D. athabasca: Alaska, Michigan, Minnesota, Ontario, Quebec. Wild-type strains except for one (Quebec-Wyoming mixed) of "bright eyes" (sex-linked recessive), several with CO_2 sensitivity.
D. azteca: California and Mexico
D. narragansett: New York
D. tolteca: Bolivia and Haiti

LOS ANGELES, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Botany

D. pseudoobscura

Lethal strains: A number of lethal strains of various gene arrangements from both Southern California and Guatemala, which are currently being tested.

Wild Strains (homozygous for third chromosome inversions)

Standard (2) San Jacinto Mountains
Arrowhead (1) San Jacinto Mountains
Arrowhead (1) Texas
Treeline (1) San Jacinto Mountains
Vandeventer (2) San Jacinto Mountains
Santa Cruz (2) San Jacinto Mountains
Pikes Peak (1) Texas
Oaxaca (1) Guatemala
Thomas Mountain (1) San Jacinto Mountains

Wild Strains carrying a lethal balanced over a marked standard

Lethal Cuernavaca (or Bl L Sc pr cv (ST)/l (CU)
Lethal Pinion Flat (or Bl L Sc pr cv (ST)/l (Pn)

Wild Strains inbred by pair matings for over 50 generations

Standard (4)
Arrowhead (3)
Chiricahua (2)

Chromosome 1

Cnv
Pt w^e
Pt mg^2
y sn v co sh
ct se ll sp tt (AR-III background)
Pt mbl y sn v - (ST-III) - XLM ST -
Pt mbl y sn v - (CH-III) - XLM CH -
or Bl L pr spr (ST)/l (CU)
or Bl L pr spr (AR)/l (CU)
or Bl L pr cv (ST)/l (CU)
or Bl L pr cv (AR)/l (CU)
or Bl L Sc pr cv (ST)/l (CU)
or Bl px^D (AR)/or L (SC)
or Bl Sc pr (ST)/or L (SC)
or L Sc pr (ST)/l (CU)
or Bl px^D (AR)/or L (SC)
or L (SC)/l (CU)
or Bl L (SC)/l (CU)
or Bl L Sc (VA)/l (CU)

Chromosome 2

gl^2
pcv^1
ubx
upt gl
upt bx Ba gl (In)l/l
upt bx Ba gl (In)/
  Dl ubx gl^2 bv
pcv ubx cd gl^2 bv

Chromosome 3

or (ST)
or (SC)
pr (AR)
pr (ST)
or px pr (AR)
or pr spr (AR)
or pr cv (ST)
or ru (TL)
or pr (ST)
or pr vg (AR)
or pr spr (ST)
or Bl L pr spr (ST)/
  or pr sp (ST)

Chromosome 4

inc j hk

Multichromosomal

gl; or pr (ST)
gl^2; or (SC)
II-III (TR or Bl L
  (SC)/l (CU)-III)

Other Species

D. hydei San Jacinto Mountains
D. tolteca Guatemala
D. biopaca Guatemala
D. simulans San Jacinto Mountains
D. melanogaster San Jacinto Mountains
D. busckii San Jacinto Mountains
D. persimilis ?

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY

D. americana americana: Independence, Ohio; Western
D. americana texana: Florida
D. ananassae: Cristobal
D. bifasciata: sex-ratio; Pavia Normal
D. busckii: Abington, Pa.
D. flavomontana: Yampa River, Colo.
D. funebris: Rexburg, Idaho; Stockholm, Sweden; Upperville, Va; white eye
D. gibberosa: South Mexico
D. hydei: Yucatan
D. lacicola: Fairbanks, Minn.
D. littoralis: Switzerland
D. melanica
D. montana: Cottonwood Canyon, Utah; LU
D. nebulosa: Haiti
D. nigromelanica
D. novamexicana
D. paramelanica
D. pseudoobscura: Pinion Standard
D. repleta: New Haven, Conn.; Philadelphia, Pa.
D. simulans
D. virilis: Japan

D. willistoni: Barbados-3; Recife-3; Recife-6; ebony; pink; white eye sex-ratio, several strains
Zaprionis vittiger: South Africa

NEW YORK, NEW YORK: COLUMBIA UNIVERSITY
(Th. Dobzhansky)

D. paulistorum

Wild Strains geographical

Bolivia (2 strains)
Brazil (5)
British Guiana
Colombia
Costa Rica (1)
Equador (3)
Guatemala (1)
Honduras (1)
Panama (3)
Peru (3)
Salvador (2)
Trinidad (4)

D. persimilis

Wild: 14 strains

Chromosome 1

se

Chromosome 2

ca
Delta ca (In)/leth

Multichromosomal

Delta or Cy

D. prosaltans

Wild: 6 strains

Chromosome 1

s m ct/fa(T1-2)
v

Chromosome 2

Bd Cy
Pm
Pm S Cy (In)/leth

Chromosome 3

ar

D. pseudoobscura

Wild Strains homozygous for different gene arrangement in third chromosome.

Mather, California
(collected in 1959)

Standard (12 strains)
Chiracahur (12)
Arrowhead (12)
Pikes Peak (12)
Tree Line (10)

Pinion, California
(collected in 1951)

Standard (13)
Chiricahua (12)
Arrowhead (10)

Texas
(collected in 1952)

Arrowhead (6)
Pikes Peak (12)

Wild Strains geographical (33)

Chromosome 1

ct se ll sp tt
Pt w^e
sex ratio/y sn v co sh

Chromosome 2

Delta gl/up gl
Delta gl (In)/leth
gl
up Ba gl
up bx Ba gl (In)/leth
up gl Tr

Chromosome 3

L or (In)/leth
or
or Bl Sc pr (In)/leth
or pr
or px
or Sc pr cv

Chromosome 4

in hk j
in hk j Cy (In)/leth
tg

Multichromosomal

Ba or Cy sprk

D. willistoni

Wild: 19 strains

Chromosome 1

se
w
w^e sn y ru

Chromosome 2

cn
Cy
abb bw
Em abb bw
S Hk abb bw (In)/leth

Chromosome 3

Delta pink (In)/leth
e
pink
ss^{ar}

Other Species

D. ambigua
D. athabasca
D. azteca (3)
D. equinoxialis (2)
D. gibberosa
D. miranda
D. mirim
D. montium
D. nebulosa
D. sturtevanti
D. tropicalis (2)

PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY

D. simulans stocks

Morro Bay
Kushla-F, Alabama
South Africa
Guatemala-3

dy f
fa rb
g bb
v
y w
net

b
bw
dh b py sd pm
net b py sd pm
stw (from su-bb)
jv se

jv st pe
pe H^h
se
st pe
ca/+ x ca

PHILADELPHIA, PENNSYLVANIA: WOMAN'S MEDICAL COLLEGE

D. robusta

40 wild strains from Ohio and Indiana
1 strain (Westchester Co., New York) homozygous for all Standard arrangements. A second strain similarly homozygous from cross of Delaware Co., Pa. female X Westchester strain males.
Washington U. stock TA (DIS 28:88).
Washington U. stock (Chadron, Neb.) homozygous for XL-1, 2L-3, 2R, 3L and 3R
1 strain (Blacksburg, Virginia) homozygous for 3L:3R-1 Ring (Genetics 37:600)

RICHMOND, VIRGINIA: MEDICAL COLLEGE OF VIRGINIA

D. ananassae: Puerto Rico (1 strain)
D. cardini: Trinidad (1)
D. equinoxialis: Brazil (1), Colombia (10), Costa Rica (10), Cuba (6), Dominican Republic (3), El Salvador (3), Guatemala (5), Hondurus (6), Jamaica (4), Panama (10), Puerto Rico (3), Trinidad (1), Venezuela (1)
D. mirim: Puerto Rico (1)
D. nebulosa: Puerto Rico (1)
D. paulistorum: Brazil (2), Colombia (1), El Salvador (7), Hondurus (1), Trinidad (17), Venezuela (1)
D. peninsularis: Puerto Rico (1)
D. prosaltans: Jamaica (1)
D. sturtevanti: Haiti (1), Trinidad (1)
D. tropicalis: Brazil (2), Colombia (20), Costa Rica (4), Cuba (11), Dominican Republic (6), El Salvador (15), Guatemala (24), Haiti (5), Hondurus (12), Jamaica (15), Panama (3), Puerto Rico (10), Venezuela (2)
D. willistoni: Argentina (10), Brazil (1), Cuba (4), Dominican Republic (5), Florida (3), Haiti (4), Jamaica (4), Puerto Rico (8), Trinidad (10)

ST. LOUIS, MISSOURI: WASHINGTON UNIVERSITY

D. robusta

Notation of arrangements as in Carson, H. L., 1953 Genetics 38:168

Stocks homozygous for gene arrangement:

Stock Designation	X	2	3	Stock Origin
Standard	SS	SS	SS	New York
BF-5	11	3S	SS	Big Fish Lake, Minnesota
TA	32	11	S1	Lake Tsala Apopka, Florida
st e-17	11	1S	SS	
C-66 (R1-C)	1S	3S	SS	Chadron, Nebraska

Miscellaneous stocks

A-1: Austin, Tex., May 1960
B-1: Bridgewater, Vt., July 1957
BF-4: Big Fish Lake, Minn. Has 3L-R balanced.
Sv-135 (R2-S): Steelville, Mo.
E-11: Eureka, Mo. Has 2L-R balanced.
Ring-3: Has ring-shaped third chromosome
Q2-35: has 2L-8 balanced.
scarletoid net: 3rd. chrom. recessives. (scarletoid formerly called "scarlet")
cardinal achrostical: 2nd. chrom. recessives.

Other Species

D. mangabeirai: obligatory thelytokous parthenogenesis
  P-29, Palmar, Costa Rica, 1959
  F-1, Frijoles, Panama Canal Zone, 1960
D. colorata: Petoskey, Mich.
D. lacertosa: UTL No. 2531.3 Sapporo, Hokkaido, Japan.
D. carsoni: Bridgewater, Vt. 1957
D. repleta: UTL 1574.1, Kilgore, Tex.
D. virilis: b tb gp cd pe
D. melanica: divers stocks
D. paramelanica: " "
D. nigromelanica " "
D. micromelanica: " "
D. euronotus: " "
D. melanura: St. Louis, Mo.

TUCSON, ARIZONA: UNIVERSITY OF ARIZONA
Department of Zoology

Cardini Group

D. cardini: s. Florida (3), Panama (2), Chile (1)
D. acutilabella: s. Florida (7), Jamaica (1).
D. cardinoides: Panama (1).
D. procardinoides: Bolivia (1).
D. belladunni: Jamaica (3).
D. dunni: Puerto Rico (2), St. Thomas (1).
D. nigrodunni complex: St. Kitts (1), Guadeloupe (1), Martinique (1), St. Lucia (1), Barbados (2).
D. similis: St. Vincent (1), Grenada (1).
D. neocardini: Brazil (1), Colombia (1).
D. neomorpha: Colombia (1), Panama (10), Honduras (1).
D. polymorpha: Trinidad (2), Colombia (2), Brazil (3).
D. parthenogenetica: Trinidad (1), Honduras (1), Nicaragua (1), Mexico (2), Colombia (1).

Local Arizona Species

D. pseudoobscura
D. macroptera
D. orbospiracula
D. arizonensis
D. azteca
D. hydei

AUSTRALIA

Melbourne, Victoria: University of Melbourne, Zoology Department

D. busckii
D. funebris
D. hydei
D. lativittata
D. serrata
D. setifemur
D. simulans

Sydney, New South Wales: University of Sydney, Department of Animal Husbandry

D. persimilis

Chromosome 1	Chromosome 2	Multichromosomal
1 se	2 cd hunchback	3 Delta or (In)/leth
		4 or Cy (In)/leth

D. pseudoobscura

Wild Strains homozygous for different gene arrangements in third chromosome:

Pinion, California
  Chiricahua (2 strains)
  Arrowhead (2)

Chromosome 2

gl

D. simulans

Wild Stock	Chromosome 1	Chromosome 3	
			st p
			net pm (b py sd)
Austin Texas	w	p	v
	y	st	

AUSTRIA

Vienna: University of Vienna, Institut f. Allgemeine Biologie

D. ambigua (strains from Austria and Scotland; 14 mutant stocks)
D. ananassae (strains from Sumatra, Recife, Brasilia)
D. azteca
D. bifasciata
D. funebris (Austria and Italy)
D. hydei (Austria)
D. miranda
D. persimilis (Sequoia)
D. pseudoobscura (Oaxaca)
D. simulans
D. subobscura (5 wild strains from different countries; inbred strains from Austria, Cambridge, Spain; about 600 inbred strains from Italy, and the Isles of Lipari, Ponza, Ventotene and Ustica; some mutant stocks)
Zaprionus ghesquierei
  " tuberculatus

Phoridae:

Puliciphora wymani (Hawaii)
Megaselia scalaris (=Aphiochaeta xanthina)
  wild strains from Napoli and Japan
  mutants: Chromosome 1: oc; Ab (T). Chromosome 2: it. Chromosome 3: D (T); Bi; sa; y; si (T); bw (T).
  different strains male-determined by Chromosome 1 or 2 or 3. (T=from Ch. Tokunaga, Kobe College, Osaka).

BELGIUM

Louvain: The University, Agricultural Institute, Laboratory of General Genetics

Wild Stocks

D. ambigua
D. americana
D. bifasciata
D. helvetica
D. novamexicana
D. obscura
D. subobscura (Belgium)
D. subobscura (Edinburgh)
D. subobscura (Pallanza)
D. subobscura (Pavia)
D. tristis
D. virilis

BRAZIL

São Paulo: Universidade de São Paulo, Faculdade de Filosofia, Ciências e Letras Departamento de Biologia Geral

D. austrosaltans: Brazil
D. cardini: Costa Rica, Mexico, Panama
D. dunni: Puerto Rico
D. emarginata: Costa Rica, Panama, and Peru
D. equinoxialis: Brazil
D. montium: Brazil
D. nebulosa: Peru and Brazil
D. neocardini itambacuriensis: Brazil
D. neocardini mourensis: Brazil
D. neocordata: Brazil
D. neomorpha: Trinidad
D. neoelliptica: Brazil
D. nigrosaltans: Panama
D. milleri: Puerto Rico
D. paranaensis pararepleta: Brazil
D. parasaltans: Brazil
D. polymorpha: Peru and Brazil
D. prosaltans: Costa Rica, Panama, Colombia, Trinidad, and Brazil
D. pseudosaltans: Brazil
D. repleta: Brazil
D. saltans: Mexico, Guatemala, El Salvador, Costa Rica, and Cuba
D. septentriosaltans: Colombia
D. simulans: Brazil
D. sturtevanti: Mexico, El Salvador, Honduras, Costa Rica, Panama, Barbados, Guadalupe, Jamaica, Sta. Lucia, Trinidad, Colombia, Venezuela, Peru, and Brazil
D. subsaltans: Brazil
D. tropicalis: Brazil
D. willistoni: Peru and Brazil

CANADA

Toronto: University of Toronto

D. simulans y w
st pe

CHILE

Santiago: Instituto de Biologia "Juan Noé" Catedra de Biologia Genetica y Evolucion Experimental

D. altiplanica: Bolivia (La Paz)
D. camaronensis: Chile (Azapa)
D. funebris: Chile (La Serena, Valdivia, Tierra del Fuego, y Punta Arenas)
D. gasici: Chile (Arica), Argentine (San Luis), Bolivia (Cochabamba)
D. gaucha: Brazil (M. Capoes, C. de Jordan and Taimbas), Argentine (Cordoba)
D. hydei: Chile (Camarones, and El Tabo)
D. immigrans: Chile (El Tabo, and Valdivia)
D. mercatorum: Chile (Arica)
D. mesophragmatica: Bolivia (La Paz), Peru (Machu-Picchu, Cuzco)
D. pavani: Chile (Copiapo, Vallenar, El Tabo, Vina del Mar, Olmué, Bellavista, Arrayan, Los Alpes, Colbum, Los Queñes, Chillan), Argentine (Mendoza)
D. serenensis: Chile (El Tabo)
D. simulans: Peru (Lima)
D. viracochi: Peru (Machu-Picchu)
D. virilis: Chile (Santiago)

COLOMBIA

Bogotá: Universidad de Los Andes

D. melanogaster (Bogotá, Mesitas, Cali, Puente Nacional, Girardot)
D. busckii (Bogotá)
D. immigrans (Bogotá, Puente Nacional)
D. hydei (Bogotá)
D. pseudoobscura (Bogotá, Guatavita, Carretera Norte)
D. pavani (Bogotá)

D. tolteca
D. sturtevanti
D. nebulosa
D. anannassae
D. latifasciaformes
(all from Mesitas del Colegio)
D. willistoni
D. emarginata
(Puente Nacional, Mesitas)
& other unidentified species

FINLAND

Turku: University of Turku, Department of Genetics

D. simulans

Wild Stock: Normal

Chromosome 3: st pe

GERMANY

Berlin-Buch: Institut für Medizin und Biologie, Genetische Abteilung
Berlin-Dahlem: Institut für Genetik der Freien Universität Berlin

D. funebris

Wild Stock	Chromosome 1	Autosome
97 Berlin-Buch	98 ev	99 st

Other Species

100 D. busckii: wild
101 D. hydei: wild
102 D. repleta: wild
103 D. simulans: v
104 D. virilis: wild

Marburg: Zoologisches Institut der Universität

D. funebris: wild

D. simulans: wild

D. virilis: $T(Y;5)pe^{m1}$

GREAT BRITAIN

London, England: University College, Department of Zoology

D. hydei: vg

D. simulans: Austin, Texas +
st pe
y w

D. subobscura

Inbred Lines	Wild Stocks
D, K and NFS (structurally homozygous) and several others	Edinburgh Galilee

Chromosome 1

Bx
m ct bnt v sc
oc
pm ct sn cp
v w/v w^{co} sc
y

Chromosome 2

ma int mj
ma mop ni
th ma mop

Standard Order

st th int ey wt
st th int mj
th ma

Inverted Order

ma mop

Chromosome 3

ho nt fd^M
+/gs

Chromosome 4

pl pp pt
sj otp pl pp

Chromosome 5

ch ar
ch cu
ch/Ba st
ch/Ba ant
ch kk^2/Ba rn

Sheffield, England: The University, Department of Genetics

D. immigrans

D. pseudoobscura: 3 strains Arrowhead gene arrangement
4 strains Chiricahua gene arrangement

INDIA

Calcutta: University of Calcutta, Department of Zoology

D. bipectinata: wild

D. simulans: wild

D. ananassae

Wild Stocks

Calcutta (a_6)+
Howrah (H_1)+

Chromosome 1

1 Cv^D
2 w
3 y
4 y w
5 ypx
6 ybo
7 ru

Chromosome 2

1 or
2 ex
3 ra'
4 ic
5 bw'
6 ss^a
7 pr
8 vs
9 ba
10 Bd
11 ss^a ra'
12 bw' vs
13 se ic
14 b ss^a
15 ra' ic
16 b ic
17 or blo
18 vs ss^a
19 bw' ss^a
20 cu ic
21 pr b
22 se cu
23 se cu b
24 ma cu b
25 pr blo b
26 pr b cu
27 se cu ic
28 se ra' ic
29 bw' cu ic
30 ex cu ic
31 pr cu ic
32 bw' vs ss^a

Chromosome 3

1 ba
2 Cy
3 pc
4 stw
5 px pc
6 stw pc
7 stw pc fu
8 stw pc px
9 fu px pc jvl
10 stw pc px fu

Chromosome 4

1 li sr dn
2 sr. dn

Multichromosomal

1 ic sr dn

ISRAEL

Jerusalem: The Hebrew University of Jerusalem

D. buzzatii
D. funebris
D. immigrans
D. lebanonensis
D. simulans: several stocks from Qiryat 'Anavim, Tel-Aviv and Jaffo
D. subobscura: several stocks containing inversions from Israel

ITALY

Milano: Universita di Milano, Istituto di Genetica

D. equinoxialis: Tefe (State of Amazones)
D. prosaltans: Cantareira (State of S. Paulo); Sangre Grand (Trinidad)
D. sellata: Cuba; Huichehugan (Mexico)
D. simulans: Aspra and Pavia (Italy). Stocks selected for tumor manifestation: tu A; tu B1; tu B3; tu C; tu Aspra
D. sturtevanti: Puerto Rico; Cantareira (State of S. Paulo)

JAPAN

Anzyo, Aichi: Nagoya University, Faculty of Agriculture

D. auraria: Anzyo-Aichi
" : Kiso
" : b (black)

D. hydei: Suginami-Tokyo
D. montium: Kanazawa
D. sordidula: Kiso

D. virilis: Nagoya
" : Tobetsu-Hokkaido
" : Tokyo

Kyoto: Kyoto University, Faculty of Science, Department of Zoology

D. ananassae
D. auraria (Type A)
D. busckii
D. rufa
D. simulans

D. virilis

Wild Stocks

1 Daisen
2 Fukushima Nihonmatsu
3 Higashiyama Hotel
4 Iida-I
5 Kanazawa
6 Niigata
7 Otsu
8 Pasadena
9 Ueda-II

Chromosome 1

10 si^{16}
11 y

Chromosome 4

12 cd

Chromosome 5

13 ru st B^{3} pe

Multichromosomal

14 ds es pe (4;5;5)
15 ru st B^{3} px pe (5;5;5;4;5)

Misima, Sizuoka-ken: National Institute of Genetics

D. americana
D. lutea
D. novamexicana 1714.4
D. texana-G
D. simulans

D. virilis

Wild Stocks

1 Fukuoka
2 Hikone
3 Kochi (Oki isl.)
4 Kuroki
5 Maruyama
6 Matsuyama
7 New York
8 Niigata
9 San Antonio

Chromosome 1

10 cv mt v^{e} sb
11 mt Bx w (2177)
12 v^{4}
13 v^{4} si^{16}
14 y ap (1047)
15 y ct ap (2507)

Chromosome 2

16 eb

Chromosome 3

17 cn

Chromosome 4

18 cd

Chromosome 5

19 es pe
20 Sb
21 st es
22 Sv po

Multichromosomal

23 si; b; t; cd; es
24 v; es pe

D. pseudoobscura

Arrowhead, Pinion Flat: 9 strains
Arrowhead, Texas: 6 strains
Arrowhead, California (1959): 10 strains
Chiricahua, Pinion Flat: 10 strains

Chiricahua, California (1959): 10 strains
Standard, Pinion Flat: 10 strains
Standard, California (1959): 9 strains
Pikes Peak, Texas: 12 strains
Pikes Peak, California (1959): 19 strains
Tree Line, California (1959): 10 strains

Osaka: Osaka University, Faculty of Medicine, Department of Genetics

D. auraria, race A (1)
D. lutea (1)
D. simulans (1)

D. virilis

Wild Stocks

1 Pasadena

Chromosome 1

2 mt w^{e} sb
3 si^{16}
4 v^{4}
5 w^{a}

6 y

Chromosome 2

7 eb

Chromosome 3

8 cn

Chromosome 4

9 cd

Chromosome 5

10 st B^{3} pe

Multichromosomal

11 v; es (1;5)
12 v; es pe (1;5)
13 cn; es (3;5)

Special Strain

14 Hikone (DDT and nicotine resistant)

Sapporo: Hokkaido University, Department of Zoology

D. auraria: Sapporo (3 wild strains), Okoppe (1), Okushiri (2), Oshoro (1)
D. lutea: Sapporo (2)
D. virilis: Sapporo (3), Shikaribetsu (1), Okushiri (2), Okinoshima (1)
D. immigrans: Sapporo (4), Okoppe (1), Kudo (1)
D. funebris: Sapporo (5), Akan (2), Ichinohashi (2), Okoppe (1), Chitose (1), Okushiri (2)
D. ezoana: Raus (1)
D. testacea: Sapporo (1)
D. brachynephros: Sapporo (2)
D. nigromaculata: Sapporo (4), Iwamizawa (1), Okushiri (2)
D. lacertosa: Sapporo (1)
D. busckii: Sapporo (2)

Tokyo: Tokyo Metropolitan University, Department of Biology

D. ananassae

Wild Stocks

1 Texas
2 TL_{1}
3 TL_{3}
4 TL_{4}
5 TL_{3-4}
6 TL_{3-11}
7 Barro Collorado, Panama 69 (Low elevation)
8 Barro Collorado, Panama 74 (" ")
9 Turrialba, Costa Rica 101 (High elevation)
10 Turrialba, Costa Rica 104 (" ")
11 Turrialba, Costa Rica 125 (" ")
12 Mampa, Africa
13 Cristobal, Panama
14 Porto Rico
15 Baton Rouge, Louisiana
16 Hawaii-H
17 Calcutta, India
18 $2L\text{-}A^{H}$
19 $2L\text{-}B^{H}$
20 2L-A; $3L\text{-}A^{H}$
21 2L-A; $3L\text{-}A^{M}$
22 $3L\text{-}A^{C104}$
23 2L-B; $3L\text{-}A^{H}$
24 2L-B; $3L\text{-}B^{H}$

Mutants

25 st f
26 ru
27 pxd
28 st f ru^{2}
29 st f se
30 y f
31 b
32 Bn-R
33 bw-R
34 fu
35 j
36 px
37 S
38 se
39 sm
40 wy

D. bifasciata

Wild Stocks

Akkeehi (3 strains)
Asakawa (1)
Gotokuji (1)
Hakkoda (2)
Kitazawa-toge (1)
Kujusan (1)
Kumotori-yama (1)
Meakan (1)
Nishitappu (1)
Okura-yama (1)
Pavia (Italy) (1)
Pfynwald (Switzerland) (1)
Shibunoyu (1)
Taisetsuzan (2)
Tanigawadake (1)

Mutants

Sex-linked

a
a y
f
y

Autosomal

ag
ar
ar ob
ar ro
arp
bn
ca ro
cn
gvh yh
ic
ic cn
M ob
ms ro
ob
or
orr
ps yh
ro
vi
yh
yh^{54}

Cytoplasmic

SR (Italy)
SR-J (3 strains)

Other Species (wild strains unless otherwise specified)

D. ambigua: Admont, Austria (1 strain)
D. americana: from America (1)
D. angularis (2)
D. auraria: A-race, wild (18), mutants (10); B-race, wild (12), mutants (2); C-race, wild (10), mutants (2)
D. bipectinata: Patan, Nepal (1)
D. busckii (6)
D. chinoi (3)
D. funebris (3)
D. hydei (5)
D. immigrans (5)
D. kikkawai (7)
D. lutea (6)
D. miranda: Big Basin (1)
D. nigromaculata (1)
D. novamexicana: from America (1)
D. obscura: Premeno, Italy (1)
D. pseudoobscura (2); mutants (1)
D. repleta: Treviso, Italy (1)
D. rufa (3)
D. simulans: from America (1)
D. suzukii (2)
D. takahashii (5)
D. tristis: Pavia, Italy (1)
D. virilis (8); mutants (6)
D. sp. of melanogaster group; from India (1)

KOREA

Seoul: Seoul National University, Department of Biology

D. auraria (2 strains)
D. bifasciata (1)
D. bizonata (1)
D. busckii (1)
D. carpiceps (1)
D. cheda (1)
D. coracina (1)
D. daruma (1)
D. grandis (1)
D. helvetica (1)
D. histrio (1)
D. immigrans (1)
D. kikkawai (1)
D. kuntzei (1)
D. lacertosa (1)
D. lutea (1)
D. magnipectinata (1)
D. nigromaculata (1)
D. puncticeps (1)
D. repleta (1)
D. sordidula (1)
D. sternopleuralis (1)
D. subtilis (1)
D. suzukii (1)
D. tenuicauda (1)
D. testacea (1)
D. transversa (3)
D. unipectinata (1)
D. virilis (1)

SOUTH AFRICA

Johannesburg: South African Institute for Medical Research

D. sp. A (see Research Notes): Ndumu Game Reserve; Pinetown; Mt. Selinda
D. sp. B: Ndumu Game Reserve; Inhaca Island
Zaprionus vittiger: Ndumu Game Reserve; Johannesburg
Z. tuberculatus: Magaliesberg

SPAIN

Madrid: Centro de Investigaciones Biológicas, Laboratorio de Genética

D. busckii: Madrid
D. funebris: Madrid
D. immigrans: Madrid
D. melanogaster: Madrid, Mallorca, Ribadeo, Rocafort, Ronda 10, Ronda 30
D. repleta: Madrid
D. subobscura: Madrid

NEW MUTANTS

D. willistoni

Report of D. F. Poulson and S. J. Counce

cvd: curved wing Counce, 59k29. Spontaneous (2females; 2 males) in reverted sex-ratio Recife-3 stock. Autosomal recessive. Males sterile, but capable of copulation with females. Females have low fertility, reduced fecundity. Viability of both sexes reduced, most mutants dying within a week of emergence. Wings are held out straight from body, and are usually curved downward, but rarely one or both may curve upward. The wing is reduced in size, but venation and proportions of cells appear normal, except at wing base which may be abnormally broad. The wing is much darker than wild type. Haltere development is affected in both sexes, but the effect of the gene is much more pronounced in females, more than half having no halteres, or only mere traces of them. In males, there is a definite reduction in the size of the halteres, although very rarely they may overlap with wild type in size, or show extreme reduction.

l^{ml}: lethal (1) mite-like Counce, 60c12. Spontaneous in reverted sex-ratio Recife-3 line. Sex-linked hemizygous lethal. Shows 50 percent crossing over with white-59f of Poulson. All three pairs of legs extremely reduced and deformed in hemizygotes, giving very mite-like appearance to males. Terminal leg segments may be fairly normal, but other segments are short, broad, twisted, and swollen. The majority of hemizygotes appear to be unable to emerge from pupal case. Those which do are extremely weak, capable only of feeble movement, and unable to stand upright. Wing expansion fails in over 90 percent of the flies which emerge. Death occurs within a few hours after emergence.

ocp: old copper Counce, 60a28. Spontaneous (9 males) in reverted sex-ratio Barbados-3 line. Sex-linked recessive. Newly emerged males have orange to red-orange eyes, females slightly redder eyes, which darken within a few hours in both sexes to a red-brown color very similar to badly tarnished copper. In very old flies, eyes may be dark brown. Viability, fertility good. Not yet tested for allelism with other known sex-linked eye color mutants. An allelic mutation occurred in a reverted sex-ratio Recife-3 stock but was lost after four generations because of low fertility and fecundity of females.

w^{59f}: white-59f Poulson, 59f. Spontaneous as single male in Recife-3 stock. Sex-linked recessive. Eyes and ocelli white, testis sheath and Malpighian tubes unpigmented. Viability good. Eye color of heterozygote duller and regularly distinguishable from wild type in crosses with Recife-3.

RESEARCH NOTES

Abrahamson, S. Reciprocal translocations induced in Drosophila oocytes.

Previous irradiation studies have indicated that a post irradiation treatment with anoxia (N_2) for a period of one hour increases the frequency of detachments of attached-X chromosomes. In the present report 3-4 day old females were irradiated with 2800 r, mated and permitted to oviposit for four days. The sons were individually tested for reciprocal translocations. The sons of females which received no nitrogen after irradiation yielded 0 translocations in 130 cultures tested. From other work, on the average fewer than 1 translocation per 1000 cultures would be expected to be induced by X-rays alone. The one hour nitrogen post-irradiation series gave 3 translocations in 644 cultures (.47%), all of which were II-III in constitution. These results are similar to those reported by Glass (1955) in which an infra-red post-treatment resulted in a .45% translocation frequency. It may therefore be suggested that nitrogen and infra-red treatments after irradiation affect the rejoining mechanism in a similar manner.

Auf der Maur, P. Uricase activity in the mutant white of *D. melanogaster*.

It could be shown that extracts from adults of the mutant white exhibit an uricase activity which is about two times higher than that of wild type. This finding explains the fact that in the body of white no uric acid or only traces of it can be found.

Baker, Rachel M. The effect of cold temperature on fertility.

For this experiment, five groups of *D. melanogaster* were used; there were approximately forty parents in each group. Group I contained untreated flies which served as the control group; Group II contained untreated males mated to females treated with a temperature of 6°C for 72 hours. The females were virgins and were mated immediately after treatment. Group III contained untreated females and males treated for 72 hours. Group IV contained both females and males which had been treated for 60 hours; Group V contained males and females treated for 80 hours.

Four five-day broods were made of each group. The number of offspring per female parent was calculated. It was found that in Group IV, there was a 25% decrease in the number of offspring from the number of offspring in the control group. Also there was a 67% decrease in the number of offspring in Group V. Notable decreases also occur in Groups II and III.

In the first broods of all five groups, no sterile flies were found. However, several flies from groups II, IV, and V showed sterility in the third and fourth broods. Far more sterilities occured in brood four of the fifth group, for only two females were still fertile by the fourth brooding. Calculations showed that by the fourth brooding, 22% of the flies that were still living were sterile in Groups II and IV; 72% of those in Group V were sterile.

	Control Group I	Untreated ♂♂ Treated ♀♀ 72 hrs. Group II	Untreated ♀♀ Treated ♂♂ 72hrs. Group III	Both ♂♂ & ♀♀ Treated 60 hrs. Group IV	Both ♂♂ & ♀♀ Treated 80 hrs. Group V
Total Offspring	1695	948	1456	964	929
Offspring per female	177.6	97.9	89.9	111.56	53.4

There were at least 20 females in each group but as some of them either became sterile or died, particularly in large numbers in groups IV and V, the number of total progeny would not seem to have a correlation to the number of offspring per female.

Barigozzi, C. and C. Halfer Abnormal appearance of inherited melanotic tumours, revealed by ovary transplantation.

During previous investigations (DIS 33) on the effect of chromosome replacements in a tumourous stock, an unexpected genetic variability arose, which seemed difficult to explain in terms of conventional genetics.

The same stock Chieti-v as chromosome donor, and stocks tu B3 and tu A2 as host have been used. Instead of replacing chromosomes, Chieti-vermilion ovaries have been implanted into tu B3 and tu A2 females, as to eliminate the doubt that the previous results might be due to incompleteness of chromosome replacement.

tu-B3 is characterized by abdominal tumours in nearly 100% of the individuals, tu A2 shows (total incidence 60-70%), in highly homozygous conditions, some thoracic tumours.

As control, a number of isogenic lines of Chieti-v have been produced, and a tumour incidence of 3% has been found.

From the ovaries implanted into the tumourous flies some eggs have been genetically analyzed. Having crossed the female carrying the supernumerary ovary to a CyL/Pm male, vermilion males have been secured and used for producing isogenic lines (25 lines from implantations into A2, and 26 from implantations into B3). Following this technique, we have excluded all eggs coming from the own ovaries of the implanted females, and we have also ruled out any possible cytoplasmic effect except for the small part of it included in the sperm.

The results obtained are the following. The isogenic lines derived from Chieti-v ovaries implanted in tu B3 females showed a very low percentage of abdominal tumours (3.9%), not exceeding the control. Out of those obtained from implantations in tu A2, three lines show as fairly high percentages (18-22%), and many tumours were located in the thorax. The character behaves as stable during 5 generations.

It is difficult to exclude a specific influence of the internal environment upon the genotype as an explanation. In fact, the difference in tumour incidence is statistically significant between the chromosomes exposed to the A2 internal environment and those exposed to that of B3; moreover, the thoracic tumours appeared only in progenies exposed to the A2 internal environment, thus providing us also with a qualitative difference between the two conditions.

It seems quite difficult, at the present time, to define what kind of influence might be exerted from the internal environment upon the chromosomes.

Further investigations are in progress to discover whether the present conclusions are still valid using other stocks as donor and as host.

Barigozzi, C. and G. Sgorbati Behaviour of the chromosomes Cy L and Pm, heterozygous with tu-B_3.

A great instability in lines isogenic for tumour factors in D. melanogaster led the authors to investigate whether isogenization was incomplete as a consequence of recombination between Cy L and Pm and tu chromosomes. This could be proved if Cy L tu, and respectively Pm tu, genotypes originate after a meiosis in heterozygous females. The results have been positive. Tumours appeared in Cy L and in Pm with an incidence of 3.7% and 4.5% respectively, which is significantly higher than the incidence in heterozygous flies (1.4 - 1.6%).

To explain which mechanism might have caused the transfer of tu-loci to the Cy L and Pm chromosomes, a preliminary attempt has been made analyzing the male meiosis since it is known that during the male meiosis crossing-over is nearly absent. Even though it is impossible to determine with the present data the recombination frequency occurring in the male, it seems that its incidence is too high (order of magnitude 10%), to be in accordance with the ordinary mechanism of crossing-over. If the interpretation holds, the recombination might be due to some other phenomenon, possibly asymmetric, and the results are, therefore, incapable of providing us with an explanation of the instability found in isogenic lines.

Beardmore, J. A. Genetic variance in relation to diurnal variation in populations of D. pseudoobscura.

An estimate of the additive genetic variance available in two sets of populations has been made. The populations are of common origin and are contemporaries, two (V) having been maintained in conditions of diurnal temperature oscillation and two (K) in constant temperature since their inception in 1956. The character used was bristle number of the fifth sternite and the heritabilities calculated were h^2 V = 0.342, h^2 K = 0.215. The difference between these figures is highly significant and suggests that the diurnal temperature oscillation used is responsible for the higher additive genetic variance of the V populations. It seems likely, therefore, that this rather short period oscillation demands that the populations within it are heterozygous to a level much above that found in diurnally stable populations.

Beardmore, J. A. Larval resistance to metallic chlorides.

Tests of wild type, se and bw strains of D. melanogaster reared on normal medium without yeast and with sodium or calcium chloride added gave survival percentages listed below.

	Percentage survival of:		
	Wild type	se	bw
on normal food	78.5	39.5	61.5
+ 1% $CaCl_2$	66.5	22.0	54.0
+ 2.5% $CaCl_2$	8.5	1.0	6.0
+ 2.0% NaCl	56.5	27.5	37.5
+ 5% NaCl	10.5	15.0	24.0

The data show (1) that the se and bw strains are relatively resistant to the higher concentration of NaCl, agreeing with the findings of Miyoshi (DIS 31, 32) and (2) that bw is also relatively resistant to $CaCl_2$, but se is not.

Belitz, H. J. The amino acids in the proteins of the pupae sheets in D. melanogaster.

For the qualitative determination of the amino acid composition of pupae sheets of the strain "Berlin wild", after washing in water, 5 and 10 sheets respectively were hydrolyzed in 6N HCl at 110°C for 15 hours. The fluid was evaporated in vacuo over NaOH. The remaining material was taken up with 80% methanol and dropped on the start points of two dimensional chromatograms. Using the solvent systems propanol: water: ammonia (70:30:1) and water saturated phenol the following amino acids were identified (arranged about in the order of increasing amounts): histidine, proline, tyrosine, aspartic acid, threonine, glycine, serine, lysine, valine, leucine, glutamic acid, and α-alanine. Besides there were present glucosamine and β-alanine in considerable amounts. On some chromatograms one or two other weak spots appeared, probably small amounts of SH-containing amino acids, which are in part destroyed by hydrolysis. The identification was done with test substances and as far as possible by specific colour reactions on the paper. Lysine and glucosamine gave two spots each.

Brink, N. G. The sterilizing action of heliotrine in D. melanogaster.

The pyrrolizidine alkaloid heliotrine has been found to affect the fertility of Canton-S males and females when it is added to the food supply of the flies. Newly emerged males and females were placed in separate vials on a sucrose-agar medium containing 0.001M heliotrine. Controls were fed on a medium unsupplemented with the alkaloid. Feeding periods ranged from 1 to 12 days. To test the fertility of the flies at the end of the feeding period, they were mated to untreated flies and the resultant progeny scored. The degree of sterility brought about was found to be variable, and treatment periods which completely sterilized all flies, also tended to reduce their longevity.

Females treated for 8 days were completely and irreversibly sterilized and their ovaries were found to be completely atrophied. (Their longevity was, however, significantly reduced, 95% of the flies dying within 10 days after treatment). On the other hand, females treated for 3 days and showing an initial 50% reduction in the number of offspring produced, generally all regained normal fertility within a week after treatment. Females treated for 6 days showed an average initial reduction of 90% in fertility, and whilst some of these flies regained normal fertility after several days, the majority remained completely or almost completely sterile. (The alkaloid only slightly reduced the longevity of 6 day treated females).

Males treated for 12 days showed an average reduction in fertility of more than 90%, but only a few of the flies were completely sterilized. (All flies treated for this length of time died within 7 days of the conclusion of the treatment). When males were mated at 2 day intervals with single virgin females, it was found that a 5 day treatment period rendered all males sterile after the first three or four broods (where there was only partial sterility), and fertility was not regained when the experiment was terminated after 3 weeks. Examination of the testes at the end of the experiment showed that the sperm tubes were completely devoid of all stages of spermatogenesis in the treated flies, but all stages were present in the controls. Since the testes contained a plentiful supply of mature sperm at the end of the treatment period, the partial sterility in the first 3 or 4 broods is probably due to the production of dominant lethals in sperm. The complete sterility observed in subsequent broods seems to be due to the destruction of the spermatogonia.

Brncic, Danko Non-random association of inversions in *D. pavani*.

The analysis of several natural populations and laboratory stocks of the neotropical species *D. pavani* Brncic 1957, has shown that there is a non-random association between gene arrangement complexes on the left and right arms of the fourth chromosome. The complexes are separated by over one-third the chromosome's length. Non-random association is observed in some natural populations and in all laboratory stocks examined.

There is a direct relationship between the number of generations a stock has been in the laboratory and the degree of association of the inversions. These data support the hypothesis that differential selection for chromosomes with certain combinations of linked arrangements is mainly responsible for this phenomenon. Apparently the existence of the two Standard arrangements or the two inversion arrangements together on the same chromosome is favored over chromosomes with the Standard on one arm and an inversion arrangement on the other.

*Brockman, H. E., J. L. Epler, and C. W. Edington** X-ray induced mosaic lethals in *D. melanogaster*.

If chromosomes of Drosophila are composed of several identical micro-fibrils or subunits, it is reasonable to assume that mutations might be induced by mutagenic agents on the subunits without affecting the entire chromosome. Such genetic changes would give rise to mosaic individuals containing cells with mutations and other cells that were normal. If both types of cells are incorporated subsequently in the gonads, mutant and non-mutant progeny would be recovered from the same parent.

Experiments have been designed to determine the frequency of X-ray induced sex-linked recessive lethal mosaics. Oregon-R males were exposed to 4000 r X-rays and mated to virgin females that were Ins(1)sc^{8L,S1R}, dl-49, y^{31d} sc$^-$ B f v/y l(1)J1 w m f. F-1 OR/Ins(1)sc^{8L,S1R}, dl-49, y^{31d} sc$^-$ B f v females were then pair mated to $\widehat{XY}$, Ins(1)EN, dl-49, y cv v f/0 males. The F-2 cultures were then examined for sex-linked recessive lethals. The non-lethal cultures were then selected, numbered, and the females that were OR/$\widehat{XY}$ in each culture were pair mated to $\widehat{XY}$/0 males. If the F-1 mother was not mosaic, her daughters should give rise to non-lethal F-3 cultures. If, however, the F-1 mother was mosaic for a sex-linked recessive lethal, some of her F-2 daughters would be heterozygous for the lethal and each F-3 culture from such females should be scored as a lethal. Other daughters of the same F-1 mother should transmit a normal X chromosome to their progeny and thus produce non-lethal cultures in the F-3.

Eighty F-2 non-lethal cultures in the experimental group have been tested for mosaics. Seventy-seven of these cultures have contained females that produce only non-lethal cultures in the F-3. Three of the F-2 non-lethal cultures, however, contained some females that were heterozygous for a sex-linked recessive lethal while their sisters transmitted X chromosomes that were free of recessive lethals. The F-1 females that produced these three non-lethal F-2 cultures were apparently mosaic for a sex-linked recessive lethal. To date no such cases have been detected in our control series.

*From Department of Biological Sciences, Florida State University, Tallahassee, Florida. Work performed under contract No. AT-(40-1)-2417 from the United States Atomic Energy Commission.

Brown, Wm. P. Some genetic parameters of egg number in selected populations of *D. melanogaster*.

Two populations of *D. melanogaster*, designated R and T, had been subjected to individual and family selection for increased egg number for 40 or more generations. Each population had attained an apparent plateau for the selected trait and crosses between them demonstrated heterosis which indicated that the populations were not genetically identical. Isogenic lines were derived to estimate genetic parameters by the analyses of Griffing (1958) for diallel crossing systems.

Intrapopulation analyses indicated that, at least for one population, additive genetic variance was nearly nonexistent while non-additive variance remained large. From interpopulation analyses the reverse was true. The heritability of egg number was estimated within each population when tested on itself. The estimates were 0.08± 0.09 for Population R and 0.30±0.12 for Population T. Also, the heritability of egg number was estimated within each population when tested on the other. These estimates were 0.23±0.09 and 0.14±0.08 for the respective populations. The latter estimates are indicative of the variation available for a scheme of reciprocal recurrent selection where selection within each population is based entirely upon the performance of crossbreds. Heritability of egg number was estimated in the closed population which was formed by crossing the two plateaued populations. This estimate was 0.19±0.06. The phenotypic standard deviation for daily egg number ranged from 18.5 to 19.8 with an average of 19.4 for all populations.

Evidence was obtained from covariance analyses for a negative genetic correlation between purebred and crossbred progenies of a common sire for egg number with estimates of -0.85 and -0.32 for the respective populations. These negative genetic correlations were supported by a decline in purebred performance during selection for improvement in crossbreds. The precise nature of this relationship remains obscure until it can be investigated more thoroughly.

(The support by the National Science Foundation and the facilities of the Population Genetics Institute, Purdue University, are gratefully acknowledged).

Burdette, Walter J. Effect of antibiotics on the frequency of lethal mutations following irradiation.

Lethal mutation rate on the X chromosome was determined in cultures of the tu 36a strain of *D. melanogaster* exposed to irradiation alone, in those treated with X irradiation and grown on medium containing actinomycin D and penicillin respectively, and in those treated with each antibiotic alone. Both antibiotics were found to diminish mutation rate following irradiation by approximately one half. The data to support this contention appear in the accompanying table.

	Control	Penicillin	Actinomycin D	Irradiation (3000r)	Penicillin + Irradiation	Actinomycin D + Irradiation
Lethals	3	8	1	155	39	31
Chromosomes Tested	2599	1150	978	2172	1036	952
Per cent	0.12	0.69	0.10	7.14	3.76	3.25

Also of interest is the mutation frequency in cultures grown on penicillin alone. In addition, the number of tumors was diminished following treatment with irradiation, actinomycin D, or penicillin. In a group of individuals of the same strain treated with N_2O during irradiation, the mutation rate was 1.34 per cent (7/521) without penicillin in the medium and 1.59 per cent (10/630) when it was present, in contrast to a rate of 5.55 per cent (27/486) following irradiation alone and 2.84 per cent (17/599) when irradiation was combined with penicillin in the culture medium. The conclusion that antibiotics are capable of inhibiting and/or reversing mutations in metazoa seems amply justified by the data presented. Apparently the mechanism is not identical with similar effects of gases. Not only do these agents offer a significant therapeutic means for altering the effects of irradiation, but also the antibiotics and congeners may be used to investigate the mechanism of genic mutation and replication.

Carlson, J. H. A new mutant stock causing an interruption of the second longitudinal vein (L_2) in *D. melanogaster.*

This mutant phenotype, arising in a wild type stock maintained in mass culture, is of particular interest since the venation effect is confined solely to the second longitudinal vein (L_2), all other veins being normal.

Chromosome substitution tests indicate that the expression of the mutant phenotype depends upon homozygosity of both the II and III chromosomes from the mutant stock. However, the replacement of a third chromosome of the mutant stock with a Hairless (H) chromosome leads to L_2 interruption, presumably through the reinforcement of subthreshold components of L_2 interruption of the H/+ and mutant II/mutant II; mutant III/+ genotypes.

Starting with an initial population of approximately 33% L_2 vein absent, it was possible, by selection at 26°C, to obtain a "high" line showing 47% L_2 absence and a "low" line characterized by an 8% average L_2 interruption. Between line matings, F_2, and backcross data suggest the presence of a polygenic system of modifiers.

Temperature studies carried out at 18°C, 26°C, and 30°C indicate a sensitivity of expression to changes in temperature. At the lowest temperature (18°C) the mean expression of the mutant stock was 11% L_2 absence, at 26°C it was 33%, and at the highest temperature used (30°C), the expression had a mean value of 41%. Over the temperature range employed, increased penetrance was accompanied by an increase in the mean amount of L_2 interruption. The pattern of expression was also affected by temperature. In general the basal segment of the L_2 vein was usually interrupted, the central section was stable, and the terminal tip showed occasional interruption. Changes in temperature altered this general pattern.

Castiglioni, M. C. and R. G. Rezzonico Tissue culture in Drosophila.

Some culture media have been experimented with with the aim of cultivating larval tissues of D. melanogaster.

Undifferentiated tissue of the larval central nervous system and cells obtained from the lymph gland seemed to us the most suitable material for our experiments.

The basic elements used in the composition of the different culture media are: plasma of heparinized cock's blood; extract of chick embryo after 8-9 day's incubation; synthetic medium for insects according to Grace; extract of D. melanogaster larvae of two wild strains. The cultures were prepared by the hanging drop method, preserved in a thermostat at 25°C±0.5 and transplanted every 7 days. The results obtained up to now may be summarized as follows:

(1) The cephalic ganglia survive in two different culture media for 4-5 weeks, although the optimal period is limited to between 14 and 21 days. In fact it is during the second week of culture that the spreading of cells outside the ganglion is noted, together with the appearance of some mitotic figures. In particular conditions (medium without plasma) numerous free cells are obtained which can be further cultivated, although isolated cells display greater requirements than the compact organ or groups of cells.

(2) The lymph gland cells can be preserved alive for 10-13 days, but no multiplication has been observed.

Chen, P. S., and C. Diem A sex specific ninhydrin-positive substance found in the paragonia of adult males of D. melanogaster.

In a previous paper Fox (1959) reported the presence of a "sex peptide" in the adult males of D. melanogaster (see also Fox, Mead, and Munyon 1959). Recent investigations in our laboratory showed that the paragonia of the male flies contain a ninhydrin-positive substance which obviously corresponds to the sex peptide reported by Fox. Two lines of evidence indicate that this substance occurs only in the paragonia of males: a) extraction of free ninhydrin-reacting-components from different organs; b) after transplantation of male genital discs into adults of the same sex, the concentration of this substance was found to increase. The fact that this substance was also found in female flies which had received male genital discs by transplantation indicates that its formation is autonomous.

Clancy, C. W. Unstained whole mounts of malpighian tubules.

In order to study the colored granules in the cells of malpighian tubules that had accummulated red eye pigment from the haemolymph, the following simple method was used for preparing permanent, unstained, whole-mounts of tubules from adult flies. The fly is dissected in a drop of insect Ringer on a slide or coverslip, preferably above a white background. When the tubes are isolated, the remainder of the fly is swept away and the excess of water removed with small, cleanly cut, strips of fine-grain filter paper. The tubules are orientated in the thin film of remaining water, and, just as the latter is about to disappear by evaporation, a drop of dioxane is applied to the tissue. Before this evaporates completely, and within one-half minute, add a drop of a 1:1 solution of dioxane-euparal. The latter solution may be changed several times by alternating removal with filter paper, and re-application. Within a few minutes, however, the excess of the dioxane-euparal solution may be removed and the mounting concentration of euparal and coverslip applied. A variation of the above procedure is to dissect the fly without Ringer using the body fluids to affix the tubes to the slide.

The microscopic appearance of tubules prepared in this manner resembles closely that observed in fresh preparations, and slides now six years old still retain characteristic differences in granule coloration noted at the time of preparation.

Clark, A. M. Mutagenic activity of dimethylnitrosamine.

In mammals, dimethylnitrosamine is a specific liver carcinogen. Tests for mutagenic activity were made by injecting 0.08 microlitres of a 10% solution in saline into the abdomen of 24 hour old Canton-S males, followed by tests for the production of sex linked recessive lethals by the Basc method. Brood intervals were 72 hours, each male being provided with 3 Basc virgin females per brood. Toxicity is rather high, only about 20% of the injected males surviving for more than two days.

Brood	Lethals	% Lethals
1	26/289	9.0
2	45/381	11.7
3	21/328	6.4
4	5/185	2.7
5	2/124	1.6

Dimethylhydrazine, a possible reduction product of dimethylnitrosamine, was tested in a similar manner but was found to be non-mutagenic.

Clutterbuck, A. J. and J. A. Beardmore Olfactory conditioning and behaviour in *D. melanogaster*.

A wild type stock was offered a choice of normal food and food containing one of the following, 3.5% peppermint oil, 7% juniper oil and 2% lavender oil, the response being judged by (1) direct counts of flies on each food, and (2) number of eggs laid on each food. In each case flies grown (adapted) for one generation and for six generations on the appropriately adulterated food were used in addition to normal flies. With the peppermint and juniper additives the adapted flies were more attracted than the non-adapted to the adulterated food by both criteria. With the lavender additive the opposite was found, i.e. the longer the period of adaptation, the greater the repulsion.

The response of two mutant stocks to lavender food has been investigated. One strain of th st cp showed a response similar to that of the wild type, the adapted flies being more repelled than normal flies by this food. In one strain of se cp e no effect of adaptation could be discerned.

The effect of adaptation producing relative distaste is remarkable, though not unique (McIndoo, Proc. Ent. Soc. Wash. 114) and has interesting implications for the theory of "biological race" formation.

di Pasquale, A. "brown spots" a character with nuclear and cytoplasmic transmission.

The investigation on brown spots has been continued (see DIS 33). To demonstrate a possible cytoplasmic activity, a cellular extract from bsp females has been injected into larvae of spotless females and males, then crossed together.

The results are the following: 1) the injected individuals failed to show spots 2) in three out of four treated stocks, spots appeared in the offspring (incidence: 0.7-7.0-11.0%), and the transmission continued.

To test whether the spots appearing in injected flies are transmitted through the cytoplasm, an F_2 has been obtained from bsp (spots induced) x Cy L/Pm. It turned out that spots are present both in heterozygotes and homozygotes, although their incidence is higher in homozygotes than in heterozygotes. The same fact results also comparing lines made isogenic for the 2nd chromosome and derived from injected individuals. For the moment, the simplest interpretation is that bsp represents a character with dual transmission, nuclear (locus on the 2nd chromosome) and cytoplasmic.

Erickson, J. and G. D. Hanks Time of temperature sensitivity in meiotic drive.

Studies were made on the temperature susceptibility of a stock resulting from the meiotic drive studies of Novitski and Hanks. It had been shown previously that the rate of X-chromosome recovery in this line is highest - 67% at 25°C, falling off to 51% at 18°C, and to 56% at 27°C.

In the present studies, males were kept at 25°C until the completion of pupation (126 hours from fertilization), then were put at 18°C until the second day of adult life. Matings were at 25°C to 15 al; ru ♀♀/♂ every two days, through five broods.

Following Khishin's analysis of the most advanced stages of spermatogenesis at various ages, the brood counts, when adjusted for temperature effect on timing, are related to meiotic stages treated as shown in the table.

		Treated-18°C		Control-25°C		Stages
Brood	Line	Total Counts	%♀	Total Counts	%♀	Stages treated at 18°C
1	B	856	69.2	1301	62.3	Meiotic divisions, spermatids, spermatozoa
	D	1238	68.0	800	65.1	
	Total	2094	68.5	2101	63.4	
2	B	1072	64.3	957	66.2	Middle primary spermatocytes to spermatids
	D	1527	66.4	997	66.4	
	Total	2599	65.5	1954	66.3	
3	B	119	57.1	359	64.6	Early primary spermatocytes to spermatids
	D	51	58.8	263	63.5	
	Total	170	57.6	621	64.2	
4	B	1026	55.0	387	65.6	Spermatogonia to meiotic divisions
	D	1147	52.0	560	64.3	
	Total	2173	53.4	947	64.8	
5	B	482	62.2	108	65.7	Spermatogonia to middle primary spermatocytes
	D	824	59.2	169	69.2	
	Total	1306	60.3	277	67.9	

Friedman, Lawrence D. X-ray induced viability mutants in *D. melanogaster*.

In order to determine both the relative frequency of X-ray induced sex-linked lethal and detrimental mutations and their effect on the viability of *D. melanogaster*, tests were made on 2520 X-chromosomes from Basc and Canton-S strains. Parents were discarded after 3 days so that the effects measured were on mature sperm.

The stock from which the experimental matings were derived was planned in such a way as to have in every generation two kinds of males, Basc and +, segregating in the same culture. In this way the X-chromosomes were controlled while the rest of the genotype was randomized. Both Canton-S and Basc males were irradiated in equal numbers. The experimental matings were as follows:

Basc/+ ♀♀ x + ♂♂ ↙
↓
Basc/+ ♀ x asc ♂♂
↓
Basc/asc, asc/+, Basc, +

Basc/+ ♀♀ x Basc ♂♂ ↙
↓
Basc/+ ♀ x asc ♂♂
↓
Basc/asc, asc/+, Basc, +

The asc chromosome is a non-B derivative of Basc and was used to detect non-disjunction.

Estimates were made on the proportions of lethals and the genetic load of lethals and detrimentals induced. A total of 1260 X-chromosomes of each type was tested at 3 different dose levels (1000, 3500, 7000 r).

(1) The induced lethal frequency compared favorably with those tested by other workers, producing an average frequency of about 2.5% per 1000 r.

(2) The ratio of the genetic load from non-lethal detrimental mutants to that from lethals was .125. The load is computed as the product of the frequency

and the average effect on viability. It is a significantly lower value than was obtained in similar studies by Kerkis and Timofèeff-Ressovsky but is in general agreement with a recent study by Käfer.

These studies also indicated that Basc males were more sensitive to irradiation than wild-type males. The lethals produced in Basc males averaged .0293 per 1000 r while those for Canton-S males resulted in an average of .0204 per 1000 r. The difference between the two strains was .0104 ± .0038 with a P value $< .05$. The Basc chromosome is therefore about 50% more responsibe to the production of X-ray induced sex-linked lethals than the Canton-S chromosome. Herskowitz and Abrahamson previously have found a similar difference with formaldehyde feeding (DIS 29) and Glass has found a difference with X-irradiation (personal communication).

Further studies on the ratio of the load due to detrimental and lethal mutants are in progress.

Fritz-Niggli, Hedi Lack of mutagenic effectiveness of sodium nitrite in *D. melanogaster*.

To prove the mutagenicity of sodium nitrite (a well known mutagen in microorganisms) in Drosophila $sc^8.Y(y^+)/y\ v$ male larvae (96 hours old) were injected with 0.01 m and 0.1 m sodium nitrite (pH 4). Immediately after emergence they were mated in two successive broods to $y^{Si}\ sc^8\ B\ f\ In^{48}\ v$; bw; e females.

The following dissection of treated males shows in 70% a deformation of the testes (lack of coiling, bulky enlargement of ellipsoidal form of testes). The offspring were tested for incidence of sex linked lethals. We found 1 lethal in 1292 chromosomes (the spontaneous mutation rate of this stock). In a second experiment we treated male gonads ($sc^8.Y(y^+)/y\ v$) of 80-hour larvae in vitro with 0.1 m NaNO2, pH 6, for 20 minutes and reimplanted the gonads in male larvae (Sevelen wild stock).

The gonads were very sensitive to the treatment and in the dissected host-flies any type of deformation can be found. Only in 2 cases did offspring result from the attached and treated testes. No lethals were detected in 76 chromosomes tested.

Fritz-Niggli, Hedi, and St. Klaas Incorporation of tritiated thymidine into different tissues of D. melanogaster.

In preliminary studies of radiation experiments we analyzed the incorporation of tritiated thymidine into the whole body of Drosophila larvae and pupae. 0 to 2-hour larvae and 48-hour larvae (after starvation for 24 hours) were fed for 4 and 24 hours with tritiated thymidine (0.05 mc tritium in 7 cc yeast-maize-food).

24 hours after feeding the 48 to 72-hour larvae we checked the uptake in the whole larvae with ionization chamber (Dynacon) and we found a mean uptake of 0.92 x 10^{-4} μc tritium. With the aid of radio-autography the distribution into different tissues was studied. We observed an uptake in the nuclei of the alimentary canal, salivary glands, Malpighian tubules, in nuclei of fat, tissue muscles, hypodermis of the nervous system cells, and in all imaginal discs.

It was interesting that we observed no uptake in the germ cells of male gonads, while the terminal cells have been labelled. The facts explain a negative mutation test.

Frydenberg, O., K. Sick, and K. Henningsen Lack of backmutations in the cn, bw and e loci.

Backmutations to wildtype in a cn; bw; e stock should be relatively easy to detect. We estimate to have screened considerably more than 100,000 cn; bw; e flies during the last two years without having found a single spontaneous backmutation in any of the three loci. Approximately 32,000 offspring of males given 3000 r (80 kV, 8 mA, 1.8 mm Al) have now been scored. No backmutations were found.

Frydenberg, O., and K. Sick Selection of st and cn.

Using artificial populations (Bennett boxes) it has been demonstrated that a mutant allele of the cn-locus and a mutant allele of the st-locus are at a disadvantage compared to the wildtype alleles. The frequency changes of the same mutant alleles have also been observed in comparable populations made homozygous for other mutants supposed to block the production of precursors to the biosynthetic steps controlled by the cn and st loci: cn versus + tested in a population homozygous for v and st versus + tested in a population homozygous for cn. Contrary to expectation, these substitutions in the genetical background did not significantly alter the rates of frequency change of the studied alleles as compared to the rates of change observed when the background was wildtype. Details to be published elsewhere.

Frydenberg, Ove , and Knud Sick
Desemination by cold shocks.

Several authors have described desemination of fertilized melanogaster females by cold shocks. We have tested the response of a number of different strains to the following treatment: the fertilized females are placed in a small metal container which is then submerged in crushed ice for 45 minutes. After the treatment the individual females are allowed to lay eggs in a standard vial for four days and their capacities to lay fertilized eggs are then judged by the number of pupal cases counted in the vial five days after the females have been removed. It has been demonstrated: 1) Different strains differ considerably in their response to the treatment. Some are very resistant, i.e. each female deposits more than 20 fertilized eggs after the treatment. Other strains are very sensitive to the treatment, i.e. a majority of the treated females yields only one or two fertilized eggs.

2) Over a period of a year or more the degree of resistance of a strain may undergo apparently spontaneous changes. In most cases the strains become more sensitive with time. This is especially true about strains recently isolated from nature. In a single case it has been observed that a laboratory strain after having been very sensitive in a year rather rapidly acquired a measurable degree of resistance.

3) Selection under brother-sister inbreeding is very effective in raising the level of resistance of a strain.

4) The degree of resistance found in a strain is largely a property of the sperm of this strain. Thus one obtains identical results when females of different strains are fertilized by the same kind of males. For this reason the following analyses of the strain differences were carried out by testing the sperm of pure and hybrid males in only one kind of female. In this way were excluded secondary variations due to varying viability and egg laying capacity of the different females.

5) An attempt to analyze the differences among strains in genetical terms has shown that hybrid sperms behave intermediately between those of the parental strains. There has, however, in all crosses been a clear difference in response between the sperms of reciprocal hybrid males. The response is always more like that of the maternal strain than that of the paternal strain. This remains true when reciprocal hybrids between an attached-X strain and a normal strain are tested. In this case the two kinds of males have identical chromosomes and the results thus indicate some sort of extra-chromosomal maternal influence.

6) Apart from the maternal effect, backcrosses have produced evidence that the response of the sperm is also influenced by the chromosomes but so far it has not been possible to interpret the chromosomal influence in any simple way. There is some indication in the data pointing to an interaction between the chromosome content and the cytoplasm.

Frye, Sara H. Corroded suppressed by extra Y-chromosome.

When males containing Corroded eye (a dominant located on the 3rd chromosome, present in stock h6, Cor/ru h D InsCXF, in the Bloomington stock list) are crossed to y f:= virgin females, the two expected phenotypic classes of females are yellow forked Corroded and yellow forked Dichaete; however, the former are only yellow, forked indicating that the expression of Corroded is suppressed at room temperature by the Y-chromosome in the female. Corroded, non-Dichaete males are obtained in the same cross.

(Work supported by a grant to H. J. Muller and associates from the U.S. Atomic Energy Commission (Contract AT (11-1)-195).)

Frye, Sara H. Failure of a carcinostatic, metabolism-requiring compound as mutagen for Drosophila.

Endoxan is the trade name for a German compound (the cyclic propanol-amidoester of bis (beta-chloroethyl) phosphamide) which releases a highly active carcinostatic principle when injected intravenously into rats, but is wholly ineffective on tumor cells in vitro. (Ref. Nature 181: 931. 1958). It was not known whether this substance is a mutagen for Drosophila and whether its mutagenic ability, too, depends on a previous metabolic transformation. If Endoxan had been mutagenic, then

Drosophila larvae would have offered a worthwhile system for the study of metabolic requirements of mutagenesis.

Feeding tests with very young larvae (0-4 hrs.) of a wild-type stock (Oregon K) on media to which various concentrations of Endoxan had been added were carried out. The concentrations used (expressed as molar concentrations x 10^{-3}) were respectively: 4, 5, 7, 9, 11, 13, & 14. The two lowest concentrations (4 & 5) slightly shortened the developmental period (i.e., young larvae to eclosed adult) while concentrations 7 & 9 slightly delayed development. The 3 highest concentrations (11, 13, & 14) resulted in a marked delay of development with few flies emerging as adults even though most larvae formed pupae which appeared perfectly normal (bristles and wings well-developed, no morphological anomalies observed) but failed to eclose. The normal males that did eclose were mated individually to Muller-5 virgin females every 5 days for 3 broods, and by the Muller-5 method the F$_2$ were scored for sex-linked recessive lethals as an index of mutagenesis. All suspected lethals were tested through another generation.

Concentration 9 yielded .52% lethals (5 lethals out of 951 chromosomes tested) while all other concentrations yielded even lower lethal percentages (percentage of lethals recovered from all concentrations in all broods was .27% (19 lethals out of 6,882 chromosomes tested). Since Endoxan requires a metabolic period of long duration for its carcinostatic effects two experimental modifications in addition to concentration 9 were made to lengthen the time of Endoxan in the system (15-30 days): first, keeping the eclosed treated males on a starvation diet for various periods (5 days and 10 days) before mating and second, paying particular attention to the late broods. It was thought that possibly a higher yield of lethals would be recovered under these modified conditions, however, the results did not bear this out. The percentage of lethals under these conditions was .44% (4 lethals out of 890 chromosomes tested) and does not differ significantly from the spontaneous frequency of lethals arising in the Oregon K stock (.17%, 4 lethals out of 2,282 chromosomes tested). Hence, under these conditions, Endoxan is not a mutagen for D. melanogaster and the study of metabolic requirements of mutagenesis with Drosophila larvae does not seem profitable. (Supported by Grant G9598 from the National Science Foundation).

Frye, Sara H. Intensity-independence of gross deletions induced by X-rays in mature sperm of Drosophila males.

Previous work (Frye, 1959), using doses of 500r and 3000r, confirmed the earlier work of Muller, Koerner, and Vogt, 1938, using doses of 1000r and 4000r on the 3/2 power relation of gross deletions and X-ray dose, thus indicating that the 2 breaks are produced by different "hits" (or separate ionization tracks), since otherwise a linear relationship between rearrangement frequency and dose would be expected to exist. In order to investigate further the production of gross deletions involving two or more breaks, a dose of 3000r was delivered to adult scute^8 Bar males (0-24 hrs. old) either continuously (total delivery time 14', intensity approximately 3.55r/sec) or protractedly (total delivery time, including intervals, of 7 hrs. 18', in thirty dose fractions of 2' 48" interrupted by time intervals of 12' 12" and at a lower intensity, approximately .6 r/sec). Thus the second treatment occupied 31 times as long a period as the first.

The treated males were mated immediately to y w In49 f virgin females (48-72 hrs. old) and allowed to mate with them for 48 hrs.; then they were allowed to mate with a second group of y w In49 f virgin females (48-72 hrs. old) for 48 hrs. The first and second post-treatment matings were designated Brood I and Brood II, respectively. These two broods represent sperm of different ages counting from the time of treatment, 0-2 and 2-4 days, respectively. Males of the expected class are y w f; those of the exceptional class are y^+ w f (hypoploid) and represent gross deletions induced in the paternal X-chromosome. All exceptional males were sterile, as expected. The results are as follows:

	Age of sperm from time of treatment					
	0 - 2 day			2 - 4 day		
Method of Delivery	y w f	y^+ w f	%	y w f	y^+ w f	% ± S. E.
Continuous	15,724	15	0.095	25,597	42	0.164 ± 0.063
Protracted	18,574	20	0.108	21,397	26	0.122 ± 0.061
Control	29,044	0	0.0	49,915	0	0.0

When protraction of an X-ray dose causes a significant decrease in gross rearrangement frequency as compared to a continuous treatment, it is usually interpreted to mean that broken ends derived from breaks produced by the earlier treatment have rejoined before those produced by the later treatment have occurred. If there had been rejoining during the 7 hour period, the protracted dose would have resulted in a lower frequency of gross deletions than the continuous dose. In the 0-2 day sperm sample the frequencies are quite similar with a very slight increase in the gross deletion frequency in the protracted dose series. There is a decrease of gross deletion frequency in the 2-4 day sperm sample with a protracted dose but it is not significant.

Taken as a whole, this work dealing with sperm in young males not exceeding an age of 4 days from time of treatment confirms earlier work dealing with mature sperm in that there is no change in multi-hit events when an X-ray dose is given all at once or spread out in time, because rejoining of breaks then induced does not occur until after fertilization.

(Support for this work was provided by a grant to I. H. Herskowitz and associates from the U.S. Atomic Energy Commission (Contract AT(11-1)-135).)

Frye, Sara H. Structure of "yellow" mutations induced by neutron treatment of scute-S1 chromosomes of mature sperm. A genetic analysis was carried out of twelve "yellow" mutants which had been induced in experiments of Muller and J. I. Valencia (1951) by exposing adult Drosophila males (2-3 days old) containing the "Binsc" chromosome (sc^{S1} B In49 sc^{8}) to a neutron dose of 1500 rads, and mating them to females containing the marker yellow. The various combinations of the respective + (indicating the normal allele) and (indicating the recessive mutant allele or deficiency) types of the loci tested are represented by the position of those marks in sequences given below. The loci, in order, are lJ1, y, ac, and bb. In addition, the symbol (l) indicates an additional lethal somewhere to the right of bb, unknown as to its position, qualitative composition and its independence of the "yellow deficiency". The numbers give the frequencies found. + - - +: 4; + - - + (l): 4; - - - + (l): 2; + - + +: 1; + - - - (l): 1 (total 12).

A comparison of the qualitative structure of neutron-induced and X-ray induced "yellow" mutants (Frye, 1958) in similar chromosomes (Binsc and $scute^{8}$) and at doses biologically equivalent (in regards to lethal-inducing effectiveness) in mature sperm suggests (since one must allow for the vast difference in the sample sizes and the long time, i.e. several years, between the recovery and analysis of the neutron-induced "yellows") that an additional lethal to the right is more likely to be associated with yellows that are neutron-induced (7/12 = 58.3%) than with those that are X-ray induced (13/108 = 12.0%). If one assumes that those additional lethals occurring where the bb locus is not affected are probably independent lethals and then compares the frequency of these y^{-} bb^{+} l (6/12 = 50.0%) that are in the neutron series with that of the corresponding y^{-} bb^{+} l (2/108 = 1.8%) cases obtained with X-rays the difference is somewhat greater. If one compares the neutron-induced y^{-} bb^{-} l (1/12 = 8.3%) with the X-ray induced y^{-} bb^{-} l (10/108 = 9.2%) there is no difference at all.

In view of the finding (Muller and Valencia, ibid.) that neutron-induced visibles at a specific locus were oftener lethal than the corresponding X-ray induced visibles (perhaps often involving 2 mutations close together but separate) it would have been of interest to determine the position of these additional lethals in order to see if they were more proximal to the yellow deficiency when neutron-induced than when X-ray-induced. This information would bear on the interpretation that the mutagenic effects of neutrons are highly localized as a result of the crowding of ionization in the proton track.

(Work supported by a grant to H. J. Muller and associates from the U. S. Atomic Energy Commission (Contract AT (11-1)-195).)

Fujio, Y. The relationship between the cephalic complexes and the eye discs in several strains of *D. melanogaster* by the tissue culture method. It is very interesting to find whether the metamorphic hormone or other substances secreted from the cephalic complexes of various strains of *D. m.* have characteristic effects for the growth and differentiation of the eye discs of these strains. To investigate this problem, the eye discs were cultured in the media containing some substances secreted from cephalic complexes which were previously cultured in rotating tube. The cephalic complexes and the eye discs used for these experiments were taken from the mature third-instar larvae of B, B(bb)-1, B(coiso), B;e^{11}(coiso), and bar-3(coiso).

The results of these experiments suggest that the cephalic complexes of B strains secrete the metamorphic hormone which has an effect upon the growth and differentiation of any eye discs, and other substance(s) - B substance - which have characteristic effects for the growth and differentiation of the eye discs of B strains.

Fung, Sui-Tong, and John W. Gowen
Sex balance in *D. melanogaster* as related to chromosome IV replication.

Our present investigation shows the IV chromosome has greater female rather than male tendencies and is similar to the X and different from the male tendencies of the other autosomes. Chromosomal determinations of the male and female intersexes from two selected triploid stocks show that their karyotypes consisted of two sets of X, three sets of II and III, with or without the Y, and having two, three, or more IV chromosomes. Male and female intersexes differed from each other in the number of IV chromosomes. Female intersexes possess two, three or more IV chromosomes while male intersexes generally have two. It appears that intersexuality in the triploids, aside from its dependence upon dosages of both the II and III chromosomes also is related to the number of IV chromosomes. The Drosophila X chromosomes have female tendencies while the autosomes are male-determining. Earlier we have reviewed other work showing that the third chromosome incorporates loci for male sex characteristics. The recent work of Krivshenko (1959) on the homology of chromosomal elements between *D. busckii* and *D. melanogaster* is of interest. His findings indicate that certain IV chromosome genes (Cell, shaven and cubitus interruptus) of *D. melanogaster* are homologous to the X chromosomal mutations of *D. busckii*. From evolutionary standpoint, the IV chromosome of *D. melanogaster* originally may have been derived from the X, and carried a portion of its female-determining tendencies with it. The female potencies of chromosome IV are rather weak requiring the 2X/3A balance to bring them out.

(This work has received assistance from Contract Number AT (11-1) 107 from the Atomic Energy Commission.)

Glass, Bentley The mutagenic effect of a 5r dose of x-rays.

Continuation of the experiment reported in preliminary status in DIS-33 has gone forward as planned. The number of individuals now scored, in control and irradiated series together, has passed the million mark. As reported last year, each parent is exposed to 5r, irradiated males are crossed with irradiated females, and dominant Minutes are scored in the F$_1$. The series are coded, so that the trained scorers do not know which series is the irradiated one. To date, 33 replications of the experiment have been carried through, and in 21 of these the Minute mutations in the treated series have exceeded the number in the control series. The cumulative results stand as follows:

Minutes in unirradiated control series	248/517,646	0.048%
Minutes in irradiated series	279/505,195	0.055%

Inasmuch as the difference between series is standing at 0.007% rather than at the initial estimate, on the basis of extrapolation from higher dosages, of 0.005%, it may not require a doubling of the present size of the data to arrive at a statistically significant conclusion. At present, a simple X^2 test yields a value, for one degree of freedom, of 2.74: $P<.10$.

It will also be noted that viability and/or fecundity appear somewhat lowered in the irradiated series, by about 2.4%.

Gottschewski, G. H. M., and W. Querner
The formation of chiasmata in the brain-eye-complex in vitro.

The eyes of insects are connected with the brain by a nervous cord crossed twice: in the chiasma internum between medulla interna and medulla externa and in the chiasma externum between medulla externa and lamina ganglionaris of the eye. In our experiments with eye imaginal discs in organ culture (see DIS 33) we were able to observe the attachment of the eye disc to the brain and the growth of the nervous cord. At the end of the 3. instar the nerve fibers are extended broad from the anlage to a small cord attached to the bulbus. At this stage twisted fibers are recognizable neither in vitro nor in histological sections. However, eye-disc-brain complexes explanted at the end of the 3rd instar and living in vitro for 24 hours, show in histological sections crossed nerve fibers in the cord. Therefore twisting of the fibers must occur in the early pupal development. At the same time the brain changes its form from round to elongated. Possible explanation: the medulla externa-part of the brain moves caudal-ventral and causes the two chiasmata mechanically at the same time.

Green, M. M. Crossing-over between y and sc.

Two additional cases of crossing over between y and sc have been found. Among the 15,747 ♂ progeny of y^2 su-w^a wa spl/y sc lz^{3N} v; Cy/+, Ubx^{130}/+ ♀♀ mated to y^2 sc w ec ♂ one y^1 su-w^a w^a spl ♂ was found. Among the progeny of 3N ♀♀ y^2 sc w^a ec/w^{aRM} (=back mutant of w^a) X FM4 ♂♂ a single y^2 w^{aRM} ♀ was recovered. Both crossovers bred true.

Hadorn, E., and H. Ursprung Xanthine-dehydrogenase in different organs of *D. melanogaster*.

According to Hadorn and Schwinck (Z. Vererb. 87: 528, 1956) implantation of wild-type Malpighian tubules and fat body into rosy larvae changes the pteridine metabolism of this mutant towards wildtype. It was assumed that wildtype tissue introduces the enzyme xanthine-dehydrogenase which is absent in ry. Various organs were therefore assayed for this enzyme, using essentially the method of Glassman and Mitchell (Genetics 44: 153, 1959). Considerable activity was detected in both larval and adult Malpighian tubules; by far higher activity was found in larval fat body. This would seem to account for the formation of isoxanthopterin in the mutant, but not necessarily for the normalisation of the drosopterin content. No activity was found in an assay on as much as 150 larval testes, whereas 80 adult testes exhibit considerable activity.

Hanks, G. D. A search for meiotic drive in natural populations of *D. melanogaster.*

Chromosomes from natural populations were tested by combining with markers on the second and third chromosomes (for example, bw and st which mark the tip of the right arm of the second and the centromere region of the third chromosome respectively). Heterozygous progeny (both males and females for each chromosome) then were tested for abnormal segregation of the major chromosomes.

A total of 64 chromosomes were tested from populations generously provided by Dr. Bruce Wallace. They had been collected in the areas: Quirath, Israel; Barcelona, Spain; Santiago Chile; and Capetown, South Africa. No promising cases were found.

Haymand, D. L., and P. A. Parsons The effect of temperature, an inversion and age of female on recombination in the X-chromosome.

The following is a summary of a paper which has been submitted for publication.

1. Recombination was studied for the three sex-linked genes v-sd-car in *D. melanogaster* using a balanced design, for the following three treatment contrasts:
   (i) 20°C and 30°C
   (ii) age of parent from 0 to 3 days, and from 4 to 7 days
   (iii) with and without the heterozygous Cy inversion

Data were collected for the eight possible treatment combinations made up of the above 3 treatment contrasts. Balanced data were obtained for each treatment combination.

2. The data collected for 3 of the 4 treatment combinations at 30°C were heterogeneous. This is due to irregular variations in recombination probably caused by developmental instability at this temperature.

3. Recombination in crosses with the Cy inversion is greater than in crosses without the Cy inversion, and recombination in the older females is less than in the younger females. At 30°C recombination is greater than at 20°C only for the sc-car segment which is proximal to the centromere. No large interactions occur between these 3 treatments.

4. Neither variation in the age of the female, nor the presence of the Cy inversion affects interference appreciably, but at 30°C, interference is considerably less severe than at 20°C.

5. The effect of temperature on recombination and interference is therefore

quite different to that of age or the Cy inversion.

Heed, W. B. Ecology and abnormal karyotypes of *D. orbospiracula*.

On April 25, 1959, 1028 *orbospiracula* and 44 *macroptera* were collected in an abandoned mine shaft, Madera Canyon, Santa Rita Mts., Arizona (5,500'). Estimated population size in the mine was from 20 to 60,000 flies. They were distributed in a non-random fashion on the roof of the tunnel in groups of 20 to 50 individuals. Sixty feet inside tunnel the temperature was 53°F and the relative humidity was 52%. In shade outside the tunnel the temperature was 70°F and the relative humidity was less than 10%. The spring of 1959 was extremely dry.

Of the sexes counted, *orbospiracula* had 506 females and 356 males; *macroptera* had 19 females and 21 males. No phenotypic abnormalities were observed in adult flies. Of 15 *orbospiracula* larval brain smears subsequently examined, two proved to have several exceptional cells (mosaic) from the typical 5 prs. of rods and one pr. of dots (male is XO).

No. Cells	Chromosome Count
4	triploid (many rods with 3 central dots)
1	tetraploid
1	8 prs. rods, 1 pr. dots
3	regular XO male but with 4 dots
1	regular XO male but with 3 dots
1	3 prs. rods, 1 pr. dots

In 1952 Heed and Wasserman collected this fungus feeder in Hidalgo, Mexico. The one larva that came through proved to be triploid in every cell examined (Clayton and Ward, 1954).

Heed, W. B., H. L. Carson, and M. S. Carson A list of flowers utilized by drosophilids in the Bogotá region of Colombia.

In August, 1960, approximately 23 species in the family Drosophilidae were collected from the flowers of at least 16 species of plants near Bogotá, Colombia. The complete life cycle of 6 species was determined either by rearing the adults directly from the flowers or by placing larvae from the flower onto drosophila medium. The purpose of this note is to attract attention to the diversity of flowers used by Drosophila as breeding sites at higher elevations in the Andes and to indicate that flowers should be investigated in other parts of the world at high elevations. Although it is well known that such flowers as *Ipomoea* and *Datura* are utilized by Drosophila, it is indeed surprising that a considerable number of Compositae were found to serve as breeding sites. Even in the city of Bogotá, Drosophila was found on various types of cultivated daisies (*Chrysanthemum*), including the small "feverfew".

The species of plants were kindly identified by Professor J. M. Idrobo, Instituto de Ciencias Naturales, Universidad Nacional de Colombia. The types of flies collected on each species of flower are listed at the end. Most of the drosophilid species are as yet undescribed and the majority of them are designated as sp. A, sp. B, etc. Certain other species were noted in the field but cannot be surely ascribed to any one of the species indicated by these letters. The number of such species is indicated in parentheses in the list.

For each of the 6 species of Drosophila of which adults later emerged, "parents" had previously been collected from that individual flower or species of flower. Such data suggest that the breeding sites of these species may be highly specific. For example, the particular species of *Zygothrica* in the list was taken only from *Salvia* flowers and was found on both the eastern and western slopes of the Cordillera Oriental.

On the other hand, there is evidence that some flowers, especially the tubular ones, may be used as a place to mate but not to breed. From previous experience it was known that certain species of *Zapriothrica* are commonly found in abundance in white *Datura* flowers but only very rarely have eggs or larvae been found in the flowers. In August, for instance, we collected 1300 individuals of *Zapriothrica dispar*, along with a few individuals each of several species of *Drosophila*, from 11 individual flowers of a single *Datura* tree. A large number of the flies were in the act of mating when the collections were made. A later count showed about twice as many males as females. Most of the decaying flowers were devoid of eggs or larvae. Eggs that were present were those of *Drosophila*, not *Zapriothrica*.

Six of the flower types in the list are yellow, 5 are red, 3 are white, 1 is purple, and 1 is white and purple. Five are zygomorphic and 11 actinomorphic, with various numbers of petals. Nine are tubular and seven are "flat". These various combinations of characters in addition to the individual scent of the flower, should provide bases for sensory discrimination by *Drosophila* of particular breeding or feeding sites.

Paramo 10-11,000 ft.

BROMELIACEAE: *Puya goudotiana* Mez - Many larvae in fermenting scape. No adults collected. No adults emerged. COMPOSITAE: *Bidens triplinervis var. macrantha* (Wedd.) Sherff - *Drosophila* sps. A and B. Eggs and larvae in flower head. Adults of A and B emerged. *Espeletia corymbosa* H. et B. - *Drosophila* sp. B. PHYTOLACCACEAE: *Phytolacca bogotensis* H.B.K. - *Scaptomyza* sp. (1). SCROPHULARIACEAE: *Digitalis purpurea* L. - *Drosophila* sp. A.

Sub-paramo 9,000 ft.

COMPOSITAE: *Bidens rubifolia* H.B.K. - *Drosophila* sps. A,B,C,D and E. *Scaptomyza* sps. (2). Larvae and pupae in flower head. Adults of C and D emerged. *Polymnia pyramidalis* Triana - *Drosophila* sp. F. Larvae in flower head. No adults emerged. *Siegesbeckia cordifolia* H.B.K. - *Drosophila* sp. G. Eggs and larvae in flower head. One larva inside ovary of a disc flower. No adults emerged. *Taraxacum officinale* Weber - *Drosophila* sp. (1). LABIATAE: *Salvia rubescens* H.B.K. - *Zygothrica* sp. Infected flowers do not open; there is only one larva per flower. No adults emerged. PASSIFLORACEAE: *Passiflora mollissima* (H.B.K.) Bailey - *Zapriothrica* sps. A and B. ROSACEAE: *Rubus bogotensis* H. et B. - *Drosophila* sps. (2). SOLANACEAE: *Datura sanguinea* R. et P. - *Laccodrosophila* sp. (1), *Scaptomyza* sp. (1).

Below Bogota 6-7,000 ft.

CONVULVULACEAE: *Ipomoea purpurea* Roth - *Drosophila* sp. H. Two larvae at base of flower tube. No adults emerged. LEGUMINOSAE: *Erythrina edulis* Posada - *Drosophila* sps. I,J and K. One egg inside fresh flower. Two larvae in decaying flowers. Seven larvae in fermenting wound of flower branch. Adults of I and J emerged. SOLANACEAE: *Datura arborea* L. - *Drosophila* sps. H, L, M and N. *Zapriothrica* sp. C. Two eggs and two larvae at base of flower tube. No adults emerged.

(The collections have been supported by a grant to Dr. Marshall R. Wheeler of the Genetics Foundation, University of Texas, from the National Science Foundation.)

Himoe, E., and G. D. Hanks A search for meiotic drive in natural populations of *D. simulans*.

Drosophila were tested for abnormal segregation in a manner similar to *D. melanogaster* (see Hanks, G. D. this issue) except that only single chromosomes were marked with mutants so that only one autosome could be tested with a single mating. Markers used were b pm (black plum) for the second; se (sepia) and st pe (scarlet peach) for the third chromosome. Altogether 38 chromosomes were tested, but 26 were tested in both sexes. Two instances were found which give counts as high as 70% st^+ in single cultures when the female is tested. The results, however, are very irregular. These cases are being investigated. The thirteen wild populations tested were obtained from the University of Texas or the California Institute of Technology.

Hiraizumi, Yuichiro, and L. Sandler Effect of enhancers of SD on the non-distorting sons of SD females.

Heterozygous SD males produce segregation ratios of 0.95 or more in favor of the SD-bearing chromosome. Heterozygous SD females always show normal, 1:1, segregation ratios. When SD is passed through a female, it may happen that half the heterozygous SD sons show no distortion although they carry the SD allele as is shown by the fact that their sons once again distort. This phenomenon is termed "conditional segregation-distortion" and is discussed in Sandler and Hiraizumi (1959, Proc. Nat. Acad. Sci. 45: 1412-1422).

It has been found that a wild stock from Tokyo, Japan (Ty) and a laboratory cn bw; e; ey^2 stock contain enhancers of SD. A test was made to see if the segregation-ratios of non-distorting SD males from SD females were affected by these enhancers. Accordingly, SD/Cy females were crossed to Ty males and to cn bw; e; ey^2 males. The resultant SD/Ty and SD/cn bw; e/+; ey^2/+ males were examined with respect to their segregation ratios. Of 48 SD/Ty males tested, 28 distorted ($\bar{k}$ = SD F_1/total F_1 = 0.97) and 20 showed almost normal segregation ($\bar{k}$ = 0.60). A *k* of 0.60 may well be due merely to viability differences. Of 53 SD/cn bw; e/+; ey^2/+ males, 35 showed a mean *k* = 0.96 and 18 gave $\bar{k}$ = 0.57.

From these results it appears that enhancers have little, if any, effect on the phenomenon of conditional segregation-distortion. A complete absence of effect, however, cannot be established from these data because (1) the mean *k* values of the non-distorting males do seem a bit high, and (2) the ratio of distorting to non-distorting males in the cn bw; e; ey^2 cross departs somewhat from the usually observed 1:1.

Hoenigsberg, H. F., A. Giavelli, L. V. Pozzi and G. P. Sironi Mutagenicity during spermatogenesis under O_2N_2 and air in *D. melanogaster*.

Using the Basc method to score recessive lethals in the F_2 and the sperm sampling method to X irradiated males the authors are studying the mutagenicity of the different stages of spermatogenesis as well as the nature of the sperm release with the method used earlier by Auerbach (Z. Vererbungslehre, 1954). So far it seems 1) that the size of sperm release is not uniform and 2) that the zone of highest mutation rate coincides with the zone of highest O_2 effect. Work in course will attempt to explain other aspects of the O_2 effect.

Hunter, Alice S. Drosophilidae of Bogotá, Colombia.

In relation to some metabolic studies of Drosophila under way in our laboratory, we have been collecting locally a variety of different species. Some of these have been definitely identified. As they may be of interest to persons working with Drosophila, a list follows. These species have been collected in a variety of ways: setting out traps of mashed fruit, sweeping fields and woods, and checking flowers for adults or larvae etc. The frequency of the different species varies with the type of habitat and method of collecting as well as temperature and rainfall, but these details will be reported on later, elsewhere.

Drosophila melanogaster, *D. simulans*, *D. busckii*, *D. immigrans*, *D. hydei*, *D. pseudoobscura*, *D. pavani*, and 2 species of the "*D. canalinea* complex" have been found in Bogotá. All stages of *D. floricola* have been found in a number of different plants. In addition we have four other unidentified species of Drosophila growing in cultures. The presence of *Zapriothrica dispar* in the flowers of Datura has already been reported on by Dr. Marshall Wheeler, and is very abundant here. Also two species of Scaptomyza have been found in Bogotá; one is *S. adusta*, and the other, a shiny black form with yellow legs, has not as yet been identified. Perhaps the only species of much interest is *D. pseudoobscura*, definitely identified by Prof. Th. Dobzhansky and Dr. Carl Epling. The author is very interested to receive cultures of flies of the *canalinea* and the *tripunctata* groups in order to compare them to local species which have not been identified.

Iyengar, Shanta V. The stability of nubbin, a vestigial-like mutant, at various temperatures.

Three groups of flies, homozygous for nubbin, a recessive gene on the third chromosome, reported by Dr. G. H. Mickey, were subjected to three different temperatures. Each group consisted of thirty parent flies, three to five days old; one group was kept at room temperature, 19°C, the second at 25°C, and the third at 28°C. The parents were removed after a five-day period and then moved to another vial. The offspring were allowed to develop at the specified temperature. They were examined when they were two days old to see if any effect of temperature was apparent in their wing development. No change in the wing length was noticed. They were examined again six days after eclosion; still no effect of temperature was seen.

Further experiments involving combinations of vestigial and nubbin are underway.

Kanehisa, T. and K. Fujita A relation between the tumor formation and xanthine-dehydrogenase activity in *D. melanogaster*.

It was strongly pointed out that a flavine (assumed to be flavine-adenine-di-nucleotide) and Mo may intimately be related to an acceleration of tumor formation itself in "melanotic tumor" of Drosophila, with antagonistic relation to iso-xanthopterine and Cu metabolisms (Kanehisa 1960a,b). It is well postulated that flavine-adenine-di-nucleotide and Mo are co-enzymes of xanthine-dehydrogenase. On the basis of these facts, to see if the enzyme activity is higher in a change of non-tumor to tumor formation, the enzyme activity of pre-third instar larva from tumor (tu, v tu) and non-tumor (wild, v) strains has been analysed, at 295mμ of Beckman-spectrophotometer, after adding of pyrophosphate buffer (pH 8.6), methylen-blue (1/2 mM), and Na-xanthine to the enzyme preparation purified as possible. The results support the above hypothesis.

Kaneko, A. A Drosophila survey made in the Sapporo Beer Product Factory.

Collections were made with fermented banana, at three outdoor places in the Sapporo Beer Product Factory, one to two times per week during a period ranging from the middle part of September to the beginning of November.

The results are summarized in Table 1. A total of 588 specimens including 11 species of the genus Drosophila was obtained. Among them, dominant species were shown by *D. melanogaster* (170 specimens), *D. virilis* (130) and *D. busckii* (116). According to Takada (unpublished), in the Nikka Factory, a whisky distillery at Yoichi, *D. funebris* and *D. virilis* were most superior in number.

It is interesting that *D. melanogaster* and *D. busckii*, which were usually less in Hokkaido, were observed as dominant species and that some species seem to show home range within this factory. Most species except *D. nigromaculata* were superior in number of males.

Table 1

Drosophila species collected in Sapporo Beer Product Factory in autumn of 1959

Species	Places			Total	
	A	B	D		
	♂/♀	♂/♀	♂/♀	♂/♀	
D. melanogaster	29/55	119/52	7/8	55/115	170
D. virilis	37/48	16/19	6/4	59/71	130
D. busckii	13/10	20/22	22/29	55/61	116
D. funebris	10/13	14/28	4/3	28/44	73
D. immigrans	4/4	12/32	1/0	17/36	53
D. lacertosa	1/3	3/4	2/5	6/12	18
D. auraria (A-type)	1/4	1/4	0/0	2/8	10
D. nigromaculata	3/1	4/0	0/1	7/2	9
D. testacea	0/0	3/5	0/0	3/5	8
D. bifasciata	0/0	1/0	0/0	1/0	1
D. lutea	0/0	0/0	1/0	1/0	1
Total flies	236	259	93		588

Kaneko, A., and T. Shima Drosophilid species collected in Okoppé and its neighboring districts in east-northern Hokkaido.

Collections were made with the use of traps and net-sweepings for a week in August, 1959. A total of 684 specimens belonging to 21 species were obtained. Among them, dominant species were represented by *D. bifasciata* (181 specimens), *D. testacea* (158) and *D. lacertosa* (103), as shown in Table 1. The results were different from those of Toyofuku and Kimura (1960) who collected in Taisei-Mura, southwestern Hokkaido, at the end of August in 1959: they observed *D. auraria*, *D. testacea*, *D. lacertosa* and *D. nigromaculata* as dominant species.

Table 1

Species	No. of flies
D. bifasciata	181
D. testacea	158
D. lacertosa	103
D. auraria (A-type)	39
D. auraria (B-type)	16
D. immigrans	37
D. nigromaculata	24
D. ezoana	21
D. suzukii	20
D. histrio	14
D. brachynephros	13
D. funebris	12
D. melanissima	11
D. unispina	6
D. tenuicauda	6
D. histrioides	5
D. busckii	1
Scaptomyza disticha	9
S. unipunctum	2
S. polygonia	1
Amiota alboguttata	1
Drosophila sp. of fenestrarum group	4
Total	684

Kang, Y. S., O. K. Chung and H. Y. Lee Seasonal changes and difference in distribution by altitude in Drosophila population.

The seasonal and altitudinal changes in distribution of three selected species of wild Drosophila populations: D. (S.) auraria, D. (D.) transversa and D. (D.) lacertosa growing at Mt. Sori in Kwangnung (Kyung-Ki province, South Korea) were surveyed from April to November in 1959. The field of observations ranged in altitude from 150 to 450 meters.

Each of selected species was found in different altitudes with no significant level.

D. (S.) auraria were found with numerical dominance in April and were found in June with less superiority. D. (D.) transversa showed their highest peak numerically in May and soon after they decreased; in August they made the lower peak again. Author could draw in June the highest peak shown D. (D.) lacertosa's distribution and then the line was down to November smoothly.

Keller, Edward C., Jr. Preliminary report on the effects of gamma irradiation on the inheritance of general viability.

Replicate lines of a long inbred strain of D. melanogaster were assayed after five generations of various treatments of selection and gamma irradiation. There were four basic types of lines: selected (for fast developmental rate) and irradiated (1000r. per generation to the eggs); non-selected (for slow to median developmental rate) and non-irradiated; selected and non-irradiated; and non-selected and non-irradiated.

Estimates of changes in the various characters are available for one of the thirty inbred replicates being tested. Assays are being made at fifth generation intervals for egg laying rate, percent pupal survival, percent adult survival, sex ratio, percent fertility, life span, and total reproductive capacity. The data presently summarized are based on approximately four pairs of parents per type of line over their entire life spans.

Table 1

Type of Line	Number of 24 Hour Replicates	$\bar{X}$ Egg Production Per 24 Hr. Period	% Pupal Survival	Estimated Total Reproductivity Per 24 Hour Period	$\bar{X}$ Length of Life in Days ♀	♂
Irradiated; selected	307	7.89	47.9%	3.54	47.2	27.0
Irradiated; non-selected	259	5.69	49.2%	2.80	51.0	24.3
Non-irradiated; selected	265	4.72	56.6%	2.67	45.8	34.3
Non-irradiated; non-selected	180	5.79	58.2%	3.37	51.8	42.2

These data indicate that the irradiated selected lines have a genotypic structure capable of producing approximately the same number of progeny as the control lines (non-irradiated, non-selected). However, in terms of the capacity for egg production there is a definite superiority of the irradiated, selected groups over all other groups.

Table 2 gives the various estimates of the various compartmented treatment effects.

Table 2

Treatments	$\bar{X}$ Egg Production Per 24 Hour Replicate	% Pupal Survival	Estimated 24 Hour Total Reproductivity	$\bar{X}$ Length of Life in Days ♀	♂
Irradiated	6.54	48.6%	3.17	49.1	25.7
Non-irradiated	5.26	57.4%	3.02	48.8	38.5
Selected	6.06	52.3%	3.11	46.5	30.9
Non-selected	5.74	53.7%	3.08	51.4	33.3

A superiority of the irradiated lines in terms of egg production is also indicated in Table 2. This increase could possibly be due to the increase in heterozygosity associated with the irradiation treatment. The irradiated non-selected and the non-irradiated selected groups were essentially similar in over all reproductivity due to the counter balancing effects of differential estimates of egg production and survival. On the average survival was reduced from 57.4% in the control groups to 48.6% in the irradiated groups. Selection for developmental rate had very little, if any, effect on survival, egg laying rate, or reproductivity; however, there was a large difference noted in the length of the female life span. Irradiation, on the other hand, had pronounced effects in decreasing male life span. Also of interest is the fact that of the initial thirty original duplicate inbred substrains used in the experiment, six irradiated groups and one non-irradiated group died as a result of sterility.

Kikkawa, H. Genetic control of amylase in D. melanogaster.

As described in DIS 32: 131, amylase activity of D. melanogaster is controlled by a semi-dominant gene located at 80± on the second chromosome. The Amy gene (strong amylase) was inserted by double crossing over method into the second chromosome having the cinnabar, brown and a weak allele of the Amy gene. The locus of the Amy gene was confirmed by using such a strain. Of interest is that even a single individual can be used as the enzyme source. Thus the character may be used as a practical material of chemical genetics in higher animals for students.

Kikkawa, H. Induced mutation of the parathion-resistant gene in D. melanogaster.

As reported in DIS 33: 142, the resistance to parathion is mainly controlled by a dominant gene located at 64.5 on the second chromosome. Any resistant strain in the world has this dominant gene. Then an attempt was performed to see whether the resistant gene is produced spontaneously or artifically from its susceptible allele. The Canton-S strain which is the most susceptible to parathion was X-rayed and the offspring were raised on the medium containing 0.5 p.p.m. parathion.

Source	Larvae tested	Survival
Control	25,000	0
2000 r	12,500	2
3000 r	16,000	2

The offspring of these resistant flies (one of them was dead before laying eggs) did not show a high resistance to parathion. However, in the course of making pure strains, one of them became very resistant. This strain showed a cross resistance to various insecticides such as DDT and BHC, but showed a negative correlation to PTU.

Koref-Santibañez, S., and O. E. del Solar Courtship and sexual isolation in *D. pavani*, Brncic and in *D. gaucha*, Jaeger and Salzano.

Courtship behavior was studied by means of the male choice method in five populations of *D. pavani* from Chile (Copiapo, in the northern part of the country, Arrayan and Viña, in the central part, Los Queñes and Chillan in the southern part of the country), in one population of *D. pavani* from Mendoza, and in two populations of *D. gaucha*, one from Brazil and another from Argentine.

It was observed that within the Chilean populations of *D. pavani* there exist homogamic courtship preferences, that is, the males used certain elements of courtship such as tappings, vibrations and lickings, preferentially toward females of their own population. Although they also spent a longer time courting these females, there was not a greater frequency of homogamic matings.

D. gaucha males exhibited one-sided mating preferences when confronted with *D. gaucha* females and with *D. pavani* from Chile.

D. pavani males from Mendoza, on the other hand, discriminated more intensely against *D. gaucha* females than did *D. pavani* males from Chile. This could be a consequence of the sympatric condition of these sibling species in Argentine.

These results indicate that within these species there exist elements which may lead to a reduction of gene exchange between different geographically isolated populations.

Lee, Taek Jun The effects of Endoxan on different stocks of wild-type *D. melanogaster*.

The larvae of the two different stocks of wild-type *D. melanogaster* were treated with Endoxan-Asta (N, N-bis-β-chloroethyl-N', O-propylene-phosphoric ester-diamide).

The larvae which were grown 70 hours were immersed in 0.5, 1, 2 or 3% solution of Endoxan respectively, and then were kept for 1 to 3 hours in the dark room. Thus treatment proved that the organs of the fly were affected in producing phenocopies. The obtained results were not related to each concentration of Endoxan and duration of treatment.

Result

1) Urbana-S	
a) Total treated number	1,200
b) Rate of hatched adults	57% of all the larvae
c) Rate of anomalies	46% of all the hatched adults
Abnormal abdomen (appeared irregular bands)	42%
Abnormal wing	4%
2) Kongju	
a) Total treated number	1,200
b) Rate of hatched adults	79% of all the larvae
c) Rate of anomalies	5.5% of all the hatched adults
Abnormal abdomen (appeared irregular bands)	5%
Abnormal wing	0.5%

The phenocopies produced most frequently and severely affected the abdomen of the fly. However, the differences of the response were conspicuous between Urbana-S and Kongju. In conclusion, it appears that there is a tendency for the two stocks to be affected differently.

Lewis, H. W., and H. S. Lewis Regulation of dopa oxidase by non-second chromosome factors in *D. m.*

The level of dopa oxidase activity in extracts of *D. m.* is controlled by at least two distinct genetic mechanisms. One involves a Mendelian locus (alpha) near the kinetochore of chromosome II. A second mechanism is responsible for variation in dopa oxidase activity in strains which carry a dominant allele at the alpha locus and has been demonstrated by means of a selection program in a Samarkand strain in which lines distinct with respect to level of dopa oxidase activity have been established. It is assumed that the high and low enzyme activity lines are similar at the alpha locus but carry different modifiers.

A similar selection program has been carried out in a Cy/Pm strain. Again high and low activity lines were readily established and the results and progress of the selection program with the balanced second chromosome strain are similar to that of the Samarkand selection program. Thus, selection for distinct levels of enzymatic activity in a strain in which the second chromosome remains intact indicates that the mechanism responsible for non-alpha heterogeneity is not linked to the alpha locus.

Evidence from preliminary experiments with a Cy/Pm; D/Sb strain crossed by homozygous recessive alpha suggests that distinct alpha alleles are carried on the Cy and Pm chromosomes respectively, and that there is a distinction between the D and Sb chromosomes with respect to modifiers.

Lüers, H. Differences in the reactions to HCN and to X-rays between a DDT-susceptible and a resistant stock of *D. melanogaster*.

Exposures to HCN of imagoes of the DDT-susceptible stock Berlin wild and of our DDT-resistant stock R II, which had been derived from the former, required nearly a doubling of time in the R II stock (10/17 min.) for killing the same percentage of individuals (75%, sex means, males more susceptible than females) under the given experimental conditions. In former experiments of Harrison (1954, DIS-28) with corresponding other stocks there existed no differences in respect to HCN-susceptibility. X-ray treatments of the males of these two stocks, individually crossed with single Muller-5 females in intervals of 3 to 3 days until the third brood, resulted in a lower number of sterile cultures, and a markedly higher number of F_1 progeny in the matings with the males of the R II stock. In two experiments with 1500r the relations in the numbers of F_1 individuals per fertile female (from a total of about 50 fertile females per brood) were 1:0.7:0.4 for the three broods of the irradiated Berlin wild males as compared to 1.8:1.2:0.5 of the DDT-resistant males. In a smaller experiment with 3000r a similar trend in these relations could be observed. Using Berlin wild females instead of Muller-5 females in another experiment with 1500r a higher yield of progeny also was registered from the DDT-resistant males. No differences in fertility can be found in the unirradiated stocks. On the other hand there resulted no striking difference between both stocks in the rates of sex-linked lethals in the mutagenicity experiments with the two doses. Further examinations of characters as fecundity, fertility, duration of life and of mutation rates shall give information to what extent there exists an intrinsic relation between lowered HCN-susceptibility and lowered radiosensitivity, separating somatic and genetic reactions.

Mange, Elaine Johansen Semistable recombinants from Baja SD.

It has been demonstrated that recombinants between SD chromosomes collected in Madison and a normal SD^+ chromosome are always less stable (i.e. show a lower mean segregation ratio and an increased variance) than the SD chromosomes collected from nature; this is due to a stabilizer, St(SD), at the tip of the SD chromosome, which is not present on the SD recombinants (Sandler and Hiraizumi, 1960, Gentics 45: 1269).

The recovery of some apparently stable recombinants from SD chromosomes collected in Baja California suggested that here a different stabilizing system might be operative (Mange, 1960 or 1961, Am. Nat.). However, the difference in tester stocks used (cn bw for Madison Sd and cn bw; e; ey or Baja SD) suggests that the cn bw; e; ey stock may carry enhancers of SD not possessed by the cn bw stock. Three generations of backcrossing a "stable" Baja recombinant (R(SD)206) to the cn bw stock has shown that the latter explanation is indeed correct: the mean segregation ratio dropped from 0.99 before backcrossing to 0.95 (258 males tested) and the range increased to include segregation ratios as low as 0.75. Since the "stable" Baja recombinant exhibits typical semistable behavior with the cn bw tester stock, it seems probable that the stabilizing system is the same in both cases.

Mather, Wharton B. Chromosomal polymorphism in *D. rubida*.

A sample of 73 *D. rubida* from Port Moresby, Goroka, Bulolo, Lae and Rabaul in Papua and New Guinea shows chromosome I to be inversion free, II L to have one simple, II R to have three simple and two complex (the complexes containing four and six inversions) and III to have four simple inversions.

The sample was fully analysed, 522 salivary squashes made from larvae from single wild females, single males mated to standard gene order females, despermed females mated to standard gene order males, and F_1 males mated to standard gene order females.

The overall inversion heterozygosity ranges from 0-5 per individual (scoring a complex as one inversion) with a mean of 1.7±1.5 and there is considerable geographical variation in gene order.

McCrady, W. B. Delayed recovery in a strain of CO_2 sensitive *D. melanogaster*

In the course of an investigation of CO_2 sensitivity in *D. melanogaster* some newly established Nebraska and Texas strains were found to be CO_2 sensitive due to a cytoplasmic factor similar to virus sigma (L'Heritier, 1958). However, one Texas strain, designated #199, was found which, after the standard exposure to CO_2 for 15 minutes at 14°C, was similar to the cytoplasmically determined sensitive strains in that all flies were completely inactive after 15 minutes following their return to air, but which differed in that many (sometimes most) of the flies began to recover soon after 15 minutes of exposure to air and were eventually able to engage in ordinary activity, including breeding, though appearing weak and less active than ordinary resistant flies. A number of tests and observations have been performed on Strain 199 along with the newly established strains with cytoplasmically determined CO_2 sensitivity, certain older CO_2 sensitive strains kindly provided from the laboratory of Dr. Ph. L'Heritier, and resistant strains.

It was difficult to determine recovery time very precisely. Nevertheless, it was found that the time required for recovery of Strain 199 flies increased with longer exposures to CO_2. Although even brief exposures killed other CO_2 sensitive strains, no mortality of Strain 199 flies was caused by exposures up to 4 minutes. None of the Strain 199 flies were killed by a second exposure to CO_2. In fact, the recovery time was greatly decreased and approached that of resistant flies. Even though flies weakened by a first CO_2 test appeared more normal in activity after a second test, offspring from such flies were affected by CO_2 exposure exactly as their parents were when first subjected to CO_2.

Flies of Strain 199 raised at 30°C almost all reacted as resistant flies upon testing and no reversions back to the sensitive state were observed when the flies were transferred to 19°C for two weeks prior to retesting. This "cure" was less pronounced when flies were raised at 26°C but the recovery time was greatly reduced below that of flies raised at 19°C. When tested at 1.5°C Strain 199 flies recovered rapidly whereas the other sensitive strains were just as susceptible at this temperature as at 14°C. Strain 199 flies did not recover at all when tested at 30°C while flies of the other sensitive strains did recover (although several hours were needed for this recovery).

Crosses have been made which indicate that this "weak" sensitivity, as evidenced by delayed recovery, is due, for the most part at least, to a single dominant gene. When males or females of Strain 199 were crossed with resistant flies, the offspring, after testing, were almost all still unconscious at 15 minutes following their exposure to air, but they recovered rapidly after this time and appeared normal by 45 minutes. The F_1 flies from both reciprocal crosses above were mated to resistant flies. In each case, approximately 50% of the offspring had recovered within 15 minutes. The remainder recovered within the next 30 minutes. F_1 X F_1 crosses were made and the testing results are given in the following table:

Cross	Exp. #	% Rec. by 15 min.	% Rec. by 45 min.	% Rec. by 1 hour	Total flies
F_1 X F_1 (199 X Resistant)	1	22.6	74.6	76.9	256
" (Resistant X 199)		30.3	84.5	85.5	517
" (199 X Resistant)	2	23.3	74.5	79.6	408
" (Resistant X 199)		27.5	77.5	78.8	690
" (199 X Resistant)	3	7.1	74.2	77.6	843
" (Resistant X 199)		22.5	76.4	78.9	1164

As shown in the table, approximately $\frac{1}{4}$ of the flies of Experiments 1 and 2 recovered after testing within the first 15 minutes, $\frac{1}{2}$ recovered within the next 30 minutes, while the remaining $\frac{1}{4}$ recovered very slowly, if at all. For some undetermined reason, the 15 minute observations in Experiment 3 vary considerably from the others but the 45 minute and 1 hour observations are very similar in the three experiments.

The balanced lethal marker stock Pm/Cy, Sb/D was used to determine which of the chromosomes contained the delayed recovery factor indicated by the above crosses. "Cy, Sb" F_1 males from the cross of Strain 199 females by Pm/Cy, Sb/D males were outcrossed to Oregon R (resistant) females. From the outcrosses, 1183 flies were classified as to "Cy" and "Sb" and were tested with CO_2. Although the results from an early test (of the first 211 flies which emerged, which were tested before the flies were over two days old) were inconclusive, two later tests involving the remaining 972 flies indicated definitely that the factor responsible for delayed recovery in Strain 199 is located on the third chromosome and has complete penetrance since none of the "Sb" flies expressed delayed recovery and all of the "non-Sb" flies did. Work is in progress to clear up the aberrant results of the early test mentioned above. Also crosses have been made to locate the factor on the third chromosome, but results are not yet available.

The possibility of an extrachromosomal factor which exerts some influence on the sensitivity phenomenon in Strain 199 is indicated by outcrosses and F_1 X F_1 crosses (as shown in the above table). The effect of exposure to CO_2, particularly seen in Experiment 3, appeared to be greater when the original "delayed recovery" parent was a female. Such a maternal effect would, of course, be expected in the presence of a cytoplasmic virus because of the presumably greater capacity of the egg for transporting cytoplasmic particles. Injections of resistant flies with Strain 199 extract are underway to determine if delayed recovery or normal sensitivity may be induced.

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Momma, E., and A. Kaneko Drosophilid flies attracted by five different yeasts.

Some ecological observations involving preference of drosophilid flies to five different yeasts were carried out in the University Botanical Garden at Sapporo during a period from May to November in 1959. The yeasts used were as follows: a, *Saccharomyces cervisiae*; b, *Hansenula anomala*; c, *Saccharomyces rouxii*; d, *Candida pelliculosa*; e, *Pichia membranaefaciens*.

A total of 23,633 specimens belonging to 32 species of the genus Drosophila were obtained with the use of 15 traps at three stations. Among them most common species were *D. auraria* (39.6% of the total flies) and *D. nigromaculata* (24.6%). Each of the most dominant species showed no significant preference to the yeasts used except yeast-e. The other species had in each their own preference to the yeasts, as shown in Table 1. It seems apparent that the more dominant species has the more extensive preference to the yeasts.

Table 1

Frequencies (%) of drosophilid flies attracted by five different yeasts. Underlines show the particularly high frequencies of the attracted flies.

Species	Yeasts					No. of flies
	a	b	c	d	e	
D. auraria	27.1	19.5	22.2	20.5	10.7	9387
D. nigromaculata	22.3	20.2	22.2	19.4	15.9	5803
D. lutea	17.9	28.1	13.5	31.3	9.2	3226
D. immigrans	11.3	35.1	11.8	28.0	13.8	2454
D. brachynephros	26.7	26.0	12.9	19.2	15.2	882
D. suzukii	20.8	28.4	14.4	22.5	13.9	564
D. lacertosa	37.0	19.9	15.1	21.3	6.7	357
D. testacea	33.2	19.2	13.0	19.8	14.8	338
D. bifasciata	16.3	36.3	11.2	21.9	14.3	251
Other 23 spp.	20.0	26.7	11.6	28.8	12.9	371
Total	22.6	23.3	18.8	22.9	12.4	23633

Moriwaki, D., and A. Fukatami Selection for sexual isolation in *D. melanogaster*.

Whether intraspecies sexual isolation would be enhanced by selection or not has been investigated by Waddington (1956) and Wallace (DIS 24: 94-96, 1950) in *D. melanogaster*. According to Waddington the continuous selections of homozygous mutant flies among the offspring raised by putting two different homozygous mutants together into a population cage, seemed to be effective increasing both homozygotes at the expense of heterozygotes every generation. Contrary to this, Wallace could not obtain such a positive result.

Concerning this problem, we made experiments, using cages and population bells (DIS 26:132) consisting of two lots, selection only and that with X-ray treatment, to examine the effectiveness of selection and on the other hand to investigate whether X-rays could enhance selection response by increasing some mutations for sexual isolation.

Two mutants, ebony and brown, of *D. melanogaster* were used in these experiments keeping backgrounds of both mutant strains nearly common. Every generation, 400 flies in total, consisting of 100 males and 100 virgin females of each strain, were put together into a cage. After having examined the relative frequencies of e, bw and wild type, only both mutant types were used as the next parents. Following the similar method as Waddington, selections of the homozygotes were continued. During about ten generations, the frequencies of heterozygotes, wild type flies, did not show significant changes, manifesting no effective response.

However, this method is not necessarily satisfactory, since possibility cannot be excluded that there coexist two classes of mutant type flies, one resulted from homogamic matings and the other from dual ones (homo- and heterogamic). Then, as another experiment, 100 mated females of each mutant type in every generation were separated individually in each vial, to examine with which males, homozygous or heterozygous, they had mated. The result presented again that the homogamic matings did not increase significantly by the successive selections.

Lastly, usual multiple choice method was adopted to estimate isolation indices to judge if sexual isolation was developed by the selection. As a result, no positive data were obtained.

In short, so far as these experiments concern, including insemination test and multiple choice technique, no effective response by the selection could be recognized.

Mukai, T., and A. B. Burdick Heterozygous effects of X-irradiation on the asymmetry of sternopleural bristle numbers in *D. melanogaster*.

Immediately after two day old isogenic males were irradiated with 0, 250, 1000, and 4000 r of X-rays respectively, they were mated to virgin females of the same strain. The degrees of asymmetry in F_1 flies which hatched on or before the 13th day after the mating were scored by the absolute difference between the sternopleural bristle numbers of the two sides. In females, the mean indices were 0.7877 (617), 0.7484 (636), 0.7348 (543), and 0.7034 (327) at 0, 250, 1000, and 4000 r, and in males 0.7863 (482), 0.7135 (520), 0.6569 (545), and 0.7597 (258) at 0, 250, 1000, and 4000 r, respectively. The numbers in parentheses show the number of flies scored. Thus, it may be concluded that asymmetry decreased as the amount of irradiation increased. Since these were isogenic flies to begin with, we can expect that the degree of heterozygosity will increase with dose. And, if the asymmetry decrease can be considered a homeostatic response (an increase in developmental homeostasis), then it should be attributed to an increase in heterozygosity, per se. One would expect that random-bred flies would not have shown this response to irradiation.

Narise, T., K. Sakai, and S. Iyama The inbreeding effect on the migrating activity of *D. melanogaster*.

The inbreeding effect on mass and random migrating activities was studied by using inbred strains derived from four natural populations; Tateba-sima (TB), Iguro-sima (IG), Te-sima (TS) and Katunuma (KN). After twenty inbred generations in TB and KN, thirty inbred generations in IG and TS populations, both random and mass migrating activities were examined for each inbred strain, sixteen strains of IG, twenty-four of TS, fourteen of KN and eleven of TB. In order to examine the mass migrating activity, 10, 20, 40, 80 and 120 flies were introduced into each original population tube and these original tubes were kept for 24 hours in a dark room at 25°C before connection with three vacant tubes. The number of flies migrating to the vacant tubes was counted 6 hours after connection. These tubes had cornmeal, molasses-agar and yeast medium. For the test of the random migrating activity, 80 flies were introduced into an original tube and the counting of number of flies migrated into the new vacant tubes was conducted in the same conditions.

1) Mass migrating activity: The variation of mass migrating activity, caused by a certain population density, was recognized among inbred strains derived from the same population and also different populations. The experimental results indicate that some strains show higher mass migrating activity and others lower one than the original population.

2) Random migrating activity: The random migrating activity, caused by random movement of individual flies, also differed from inbred strain to inbred strain. Significant differences between populations were found. From the results of the experiments, it can be said that some strains show higher random migrating activity and others show lower than that of the original population. The interesting fact is that the mean values of mass as well as random migrating activity of inbred strains from one population are not so different from that of the original population. These findings indicate that the inbreeding does not bring about the inbreeding depression regarding the migrating activity and two kinds of migrating activities should be controlled by genetic factors.

Nawa, S. The structure of the yellow pigment from Drosophila.

The yellow pigment (sepiapterin), occurring in the eyes of the mutant sepia, decomposed to one mole each of 7,8-dihydroxanthopterin and lactic acid accompanied by consumption of 0.5 moles of oxygen, upon shaking its aqueous solution in air under the presence of borax. The pigment, in sodium bicarbonate solution, consumed one mole of periodate to give acetaldehyde and 2-amino-4-hydroxy-7,8-dihydropteridine-6-carboxylic acid. These results suggest that the compound is a 7,8-dihydro-form of 2-amino-4-hydroxy-6-lactylpteridine.

Nicoletti, B., and D. L. Lindsley Cytogenetic analysis of T(X;Y)'s.

We have in press in Genetics a cytogenetic analysis of 87 cases of induced linkage between the X and Y chromosomes. Space limitations did not permit publication of the breakpoints of each translocation. These breakpoints have been tabulated and are presented here to supplement the paper in Genetics. The translocations are grouped according to phenotype as described in the first column of the table. The phenotype is defined according to four characteristics: T(X;Y)/Y viability, T(X;Y)/Y fertility, T(X;Y)/0 viability, and T(X;Y)/0 fertility respectively. + indicates viable or fertile; - indicates lethal or sterile; and o indicates not tested or not testable. Where a strain carries two independent rearrangements, they are described on two successive lines in the table. Translocations between the X and two different Y chromosomes have been recovered: $y^{+}Y = sc^{8} \cdot Y$ and $B^{S}Y$.

Cytological Constitution

			Breakpoint in			
Phenotype	No.	Y used	Y	X	Autosome	Remarks
++++	148	BS	L	2D		
	140	BS	L	3C		
	104	BS	L	3D		
	8	y+	L	4B		
	149	BS	L	6E		
	4	y+	L	11A		
	129	BS	L	11A		
	125	BS	L	15D		
	159	BS	L	18A		
	2	y+	S	5E; 11F; 19F		$1A-5E/Y^{P}; Y^{D}/11F-19F/5E-11F/X^{P}$
	131	BS	S	6E		
	147	BS	S	8F		
	169	BS	S	11D		
	151	BS	S	19F		

Cytological Constitution

Pheno-type	No.	Y used	Breakpoint in			Remarks
			Y	X	Auto-some	
+++-	107	BS	L	3C		
	10	y+	L	3E		
	102	BS	L	7D		
	156	BS	L	7D		
	124	BS	L	9F		
	152	BS	L	13A		
	142	BS	L	13E		
	240	BS	L	14A		
	157	BS	L	14F		
	106	BS	L	16A		
	118	BS	L	16E		
	19	y+	L	17A		
	117	BS	L	17A		
	135	BS	L	18C		
	22	y+	S	19E		
	101	BS	S	19E		
	133	BS	S	19E		
	141	BS	S	19E		
	11	y+	S	19F		
	14	y+	S	19F		
	18	y+	S	19F		
	105	BS	S	19F		
	119	BS	S	19F		
	123	BS	S	19F		
	132	BS	S	19F		
	137	BS	S	19F		
	113	BS	S	20A		
	122	BS	S	20A		
	139	BS	S	20A		
+-+o	21	y+	L	1F		
	109	BS	L	3C	40-41	2 linkage genetically determined
	136*	BS	L	7A	70C	Y^DX^P; A^DY^P; X^DA^P
	17	y+	L	7B	39	Y^DX^P; A^DY^P; X^DA^P
	12	y+	L	7B	50-51	Y^DX^P; A^DY^P; X^DA^P
	146	BS	L	7D		T(X;Y) +
				20A	57F	T(X;2)
	20	y+	L	11A		
	138	BS	L	11A	84B	Y^DX^P; A^DY^P; X^DA^P
	5	y+	L	11D		T(X;Y) +
				14F	72	T(X;2)
	130	BS	L	11F	40-41	2 linkage genetically determined
	1	y+	L	16F		
	9*	y+	S	2C; 19F		1A-2C/Y^P; Y^D/19F-2C/19F-20
	150	BS	S	3F		
	154	BS	S	10A	97A	Y^DS^P; A^DY^P; X^DA^P
	145	BS	S	11B		
	134	BS	S	12E		T(X;Y) +
				19E	62A	T(X;3)
	116	BS	S	14D		T(X;Y) +
				9C	101	T(X;4)
	160	BS	S	17C	40-41	2 linkage genetically determined
	110**	BS		19D	55F	T(X;2) +
			S		45F	T(Y;2)
	127	BS	S	19F	85E	Y^DX^P; A^DY^P; X^DA^P
+--o	143	BS	L	12A		T(X;Y) +
				3F	69C	T(X;3)

Cytological Constitution

Phenotype	No.	Y used	Breakpoint in			Remarks
			Y	X	Autosome	
+--o	144	BS	L	15E	74D	Y^DX^P; A^DY^P; X^DA^P
-ooo	111	BS	L	3C		
	114	BS	L	3C		
	128	BS	L	3C		
	164	BS	L	3C		
	16	y+	L	4C		
	108	BS	L	5D		
	13	y+	L	7D		
	158	BS	L	11A		
	15	y+	L	14F		
	112	BS	L	15A		
	161	BS	L	17A	94	Y^DX^P; A^DY^P; X^DA^P
	290*	BS	S	1A; 20A		$1A/Y^P$; $Y^D/20A-1A/20$
	3	y+	S	3E		
	121	BS	S	6F	86D	A^DX^P; X^DY^P; Y^DA^P
	155	BS	S	7B		
	115	BS	S	8F		
	6	y+	S	11D		
	100	BS	S	13F		
	7	y+	S	14F		T(X;Y) +
			L		36C	T(Y;2)
	153	BS	S	17A	35D	Y^DX^P; A^DY^P; X^DA^P
	120*	BS	S	17E		
	103	BS	S	19F		

* lost

** the only case in which X;Y linkage was not accompanied by X;Y interchange.

Novitski, E. Correction of a note in DIS-30.

The note on page 143 of DIS-30 entitled "The possibility of another cytoplasmically inherited factor in D. melanogaster" has proved to be in error. The phenomenon described therein, the appearance of only female progeny from certain crosses, have since been shown to result from (1) the use, in the male, of a poorly transmissible X chromosome, (2) the absence of a free Y chromosome in certain stocks used as one parent coincidentally with the absence of the bb locus on the X chromosome of the other parent, and (3) the existence of several cultures which produced only female offspring for neither of the above reasons, and still not explained, but which proved to be unrepeatable. It is calculated that the probability that these three factors, each moderately unusual by itself, would appear simultaneously in a single experiment in such a way as to suggest a completely self-consistent but unusual (and also incorrect) explanation is so low as to suggest the operation of some factor other than chance.

Ogaki, M. Effect of feeding lactamide on the facet number of double Infrabar and translocation Bar strains in D. melanogaster.

Feeding experiments of some chemicals at critical period during larval life forked double Infrabar, $T(1;4)B^S$ and of a new Bar mutant known as $T(1;3)B^{581}$, which was placed at the author's disposal by E. B. Lewis, indicate that lactamide is the most effective one as a facet-increasing substance to all of these mutants. In females of B^{581} and B^S and in males of f B^iB^i the facet number increase almost to the point of normal facet number of feeding of 2.5 per cent of lactamide in the normal culture media.

Ogita, Z. Genetical and biochemical studies on negatively correlated cross-resistance in D. melanogaster.

The cross-resistance pattern of DDT, BHC, parathion and phenylurea is negatively correlated with phenylthiourea, while the pattern of DDT, BHC and parathion is positively correlated with phenylurea.

To understand the biochemical mechanism of the negatively correlated cross-resistance, and to find substances having toxicities stronger than phenylthiourea, the author has investigated the relationship between structure and negatively correlated activity in a number of compounds, using genetical analyses.

The results indicated that the p-halogen substituents of phenylthiourea were also negatively correlated to DDT, BHC, parathion and phenylurea, and that the p-halogen substituents of phenylurea were positively correlated to DDT, BHC and parathion. These facts affirmed the suggestion that phenylthiourea analogues are clearly negatively correlated substances whereas these corresponding oxides (phenylurea analogues) are positively correlated.

The addition of substituents into the thioureido radicals of phenylthioureas reduced their toxicity, and resistance to these chemicals was not negatively correlated with general insecticides.

Certain other thiourea derivatives tested failed to show a negative correlation with DDT, BHC and parathion, and most of them seemed to be positively correlated with nicotine sulfate.

Okada, Toyohi A proposal for "Burla's rule" to the relation seen between the lengths of the wing-veins and of the wing itself in the drosophilid flies, and its analysis in view of allomorphosis.

It was discovered by Sturtevant (1942) that among various species of Drosophila the costal index is negatively correlated with the 4th-vein index, and Burla (1954) found that the costal index is positively correlated with, and the 4th-vein index and the 4C-index are negatively correlated with, the wing length among a series of species of the African Pholadoris. The same relation has been proved by the author (1959) to be accepted not only by the systematic categories higher than species (subgenera, species-groups, sibling species, etc.) but also by those lower than species (subspecies, races, strains, individuals, etc.), and it was named "Burla's rule". The relation was analyzed by the author (1960) from the standpoint of allomorphosis between the lengths of the second section of costa and of the wing itself, which resulted in the finding that the allometric constant among the species having smaller wings is less than 1 (e.g. 0.85 in Sophophora and Paradrosophila) while it attains nearly 1 or isometric in the species having larger wings (e.g. 0.98 in the subgenus Drosophila). Moreover, the intraspecific allometric constant was proved to be larger in the species having larger wings, eventually fit to the rule of Lameere and Geoffrey Smith.

Okada, Toyohi The larval mouth-hooks of the family Drosophilidae, in relation to the systematics and the feeding habits.

The larval mouth-hooks (mandibles) of about 50 species of Japanese Drosophilidae are divisible into four major types - type A without teeth, type B with one tooth, type C with two to several teeth, and type A' with numerous (about ten to twenty) minute teeth. With a few exceptions, the first instar larvae show the types A or B, the second instar larvae the type C, while the mouth-hooks of the third instar larvae differentiate into various types roughly correlated with the systematic positions and the larval feeding habits. For example, the species of Amiota, Drosophila (Paradrosophila), and some of the virilis section of the subgenus Drosophila, which are thought to be comparatively "primitive" among the family and are mostly sap-feeders, show the type A predominantly, the species of Drosophila (Sophophora), which feed on fruits, show the type A', and the species of Mycodrosophila, Drosophila (Hirtodrosophila), and most of the Quinaria section of the subgenus Drosophila, which are thought to be more "advanced" and are fungus-feeders, have the type C in general. It will be suggested that the types of the first and the second instars are rather intrinsic in nature, characteristic to the respective instars as a whole, while those of the third instars are apt to be influenced by the extrinsic environments, such as the food substances.

Oshima, C. Persistence of lethal chromosomes in experimental populations.

The heterozygous male and female flies (ten pairs) having induced lethal and normal Samarkand second chromosomes were introduced into a vial and the population was transferred into a new vial twice a week. The experimental population has been continuously cultured in six vials. This culturing method is the similar one used by

A. A. Buzzati-Traverso (1947).

From the starting date, generations were counted by the intervals of fifteen days. The number of flies in a population increased gradually and after a few generations about 800 flies have been cultured in the six vials. Four populations were replicated by using every lethal strain. The genetic background of flies was presumed to be isogenic except for an irradiated second lethal chromosome at the initial population, but it would become somewhat heterozygous by spontaneous mutations after the following generations. The zygotic frequencies of lethal chromosomes in six different experimental populations, which have different lethal chromosomes individually, have been tested in the tenth, twentieth and twenty-sixth generations. The number of sampled chromosomes from each population was about one hundred. The mean lethal heterozygote frequencies of these six populations have decreased from 1.0000 to 0.1509, 0.0967 and 0.0219 respectively. When these changing lethal heterozygote frequencies were compared with the theoretical frequencies ($s=0$), there were no significant differences between them.

On the other hand, the experimental populations initiated by flies having lethal and normal second chromosomes extracted from natural populations, have been cultured by the same method described above. The X and third chromosomes were isogenic Samarkand chromosomes. The changing frequencies of lethal chromosomes in the third, sixth, and ninth generations were tested by sampling about one hundred chromosomes from five different experimental populations. The mean lethal heterozygote frequencies in these generations were estimated as 0.6236, 0.5586 and 0.5486 respectively. These frequencies are much higher than the theoretical frequencies ($s=0$). The remarkable difference between both experimental results would be ascribed to the effect of their genetic backgrounds resulting from natural selection.

Oshima, C., and O. Kitagawa Frequencies of second chromosomes, having deleterious effects on viability when homozygous in natural populations of *D. melanogaster*.

Two hundred and eighty-two second chromosomes were isolated from five small and four large natural populations by CMI method. When they were duplicated, their deleterious effects on viability were observed. The other main chromosomes were substituted by Samarkand isogenic chromosomes. The frequencies of lethal, semi-lethal and deleterious chromosomes in 134 second chromosomes extracted from small populations were observed to be 11.94, 5.97 and 14.93 percent respectively.

On the other hand, the frequencies of above mentioned chromosomes in 148 second chromosomes extracted from large populations were observed to be 14.19, 18.24 and 16.89 percent respectively. From this result, it can be concluded that the large populations maintained more deleterious chromosomes, especially semi-lethal chromosomes than the small populations.

One hundred and eighty-five second chromosomes were isolated from the same small populations at the similar time of this year. The frequencies of lethal, semi-lethal and deleterious chromosomes were observed to be 12.97, 2.16 and 8.11 percent respectively. The frequencies of deleterious chromosomes were not markedly fluctuating.

Oshima, C., and O. Kitagawa The pre-adult viability of heterozygotes of *D. melanogaster* second chromosomes that are lethal and semi-lethal when homozygous.

The effects of X-ray induced lethal second chromosomes on viability in heterozygous condition were reported in DIS-33. The mean coefficient of selection ($\bar{s}$) of these twenty-seven induced lethal heterozygotes was estimated to be 0.04636, but their coefficients of selection were found to be greatly influenced by their genetic backgrounds. If the lethal free genome was substituted by an unrelated genome extracted from an experimental population, the mean coefficient of selection was raised to 0.01640.

The lethal and semi-lethal second chromosomes were isolated by CMI method from nine natural populations in October, 1959. The X and third chromosomes were constituted with lethal, semi-lethal and normal chromosomes respectively. The mean coefficients of selection of thirty-five natural lethal heterozygotes and thirty-one semi-lethal heterozygotes were estimated to be 0.01243 and 0.00678 respectively.

Parsons, P. A. Weight and sterno-pleural chaeta number in *D. melanogaster*.

Parsons and Kroman (Heredity, 15:301, 1960) observed that phenyl-thio-carbamide (PTC) kills ebony larvae at much lower concentrations than wild type larvae, since it is a tyrosinase inhibitor, the tyrosinase content of ebony larvae being considerably lower than in wild type larvae. (Wolsky and Kalicki, Nature, 183:1129, 1959). PTC reduces the size of the adult, and delays emergence in sub-lethal doses. A study has been recently carried out on the correlation between sterno-pleural chaeta number and body weight, at two levels of competition (25 and 100 larvae per vial of 8 gms. of medium) on various concentrations of PTC for e"e", e"+ and ++ (Oregon-R) genotypes.

Both weight and chaeta number decrease with increasing PTC concentration. At the high level of competition, weight and chaeta number are lower than at the low level for all PTC concentrations, as might be expected. The range of body weights was from 1.30 to 0.25 mgm. and chaeta number from 24 to 13.

Preliminary results indicate linear relations between chaeta number and body weight within each genotype irrespective of treatment or level of competition. If x is the body weight in mgm., and y the chaeta number, approximate relations between body weight and chaeta number are approximately:

$$\begin{aligned} ++ \; &; \; y = 12.37 + 6.8x \\ e''+ \; &; \; y = 12.93 + 5.7x \\ e''e'' \; &; \; y = 12.40 + 7.5x \end{aligned}$$

Thus slopes of the lines relating x and y differ somewhat between genotypes. In particular, the chaeta number of e"+ decreases relatively less with decreasing weight than the two homozygotes. Furthermore, e"+ genotypes survive higher PTC concentrations than ++, and also survive at lower weights than ++ and e"e". Perhaps the lower rate at which bristle number decreases in e"+ flies is a consequence of somewhat better developmental stability of e"+ than the homozygotes.

It may be expected that chaeta number is a reflection of body area, since chaetae are apparently largely a surface phenomenon. However, from the above equations, at zero body weight, 12 to 13 chaetae per fly would be present, showing that chaeta number decreases much more slowly than surface area.

Paterson, H. E. Sibling species in the melanogaster species-group in Africa.

Burla (DIS-27, 1954a & b) has referred to *D. seguyi* Smart, discussing the species relationships with other members of the montium subgroup, and mentioning a sex-limited colour polymorphism in the species.

A survey was recently carried out to determine how abundant and widespread *D. seguyi* is in the eastern half of Southern Africa, and to decide whether or not it is a suitable species for population-genetic study. On examination of the material collected, it was noticed that the genital arch, forceps and anal plate separated two distinct sub-groups within the group which had previously been referred to as *D. seguyi*. The more abundant and widespread subgroup has a genital arch and hypandrium corresponding with figures 3 and 6 in Burla's 1954b paper (Rev. Brasil Biol. 14(1): p. 45); the genital arch in the other subgroup corresponds with figure 162 in Burla's 1954a paper (Rev. Suisse Zool. 61, fasc. suppl., p. 158).

That we are dealing with two sibling species is clear from the following evidence: (1) the morphological differences separating the males of the two groups are striking when once noticed (2) the forms are often sympatric (3) sexual isolation between them appears to be complete, as judged by unsuccessful attempts to cross them in both directions. Until it is possible to examine the type it will not be possible to decide which form represents D. seguyi. In the meantime, the species with the genital arch figured by Burla in his 1945a paper (fig. 162) will be called Species A and the species, the genital arch and hypandrium of which Burla figured in his 1954b paper (figures 3 & 6), Species B.

At my request, Professor Burla has very kindly examined the five males mentioned on page 160 of 1954a paper and informs me that two of the specimens belong to species B, and the rest to species A, if the characters of the genital arch, forceps and anal plate are used. However, if the characters of the hypandrium and anterior parameres are considered the three specimens assigned to species A fall into two groups. A single male is of the form found in Southern Africa, having the same type of hypandrium and with the anterior parameres long, while the other two specimens have hypandria of a somewhat different form and short anterior parameres. It thus seems possible that there are three sibling species in this complex. A decision on the status of the third form will have to await the obtaining of laboratory cultures of the form.

The female-limited colour polymorphism mentioned by Burla (DIS-27) occurs in both species A and species B.

Petit, C., and M. Guillaumin The influence of breeding on the homogeneity of populations.

Population geneticists are always coming up against the problem of representativity of laboratory stocks; so we have tried to assess the influence of breeding on the homogeneity of stocks.

Ten lines have been made from a heterogeneous population of 2000 flies. Five lines were produced from five pairs taken at random; these lines were continued with every generation by one pair taken at random in each line. There were five other lines, each of which came from four pairs taken at random and bred in the same way at every generation.

The character we have chosen, namely the number of ovarioles, was used to measure the homogeneity of the populations, using the right-left correlation coefficient given by Reeve and Robertson (1954).

When isolated, the lines Nos. 1, 2 and 3, coming from one pair are heterogeneous. The lines 4 and 5 have a correlation coefficient just significant for 0.05 probability, so it is not certain that these lines are homogeneous. After twenty five generations of brother and sister mating, the five lines are homogeneous; besides lines 1, 2, 5 are quite stable, as to the average number of ovarioles, whereas lines 3 and 4, kept with great difficulty on account of long periods of semi-sterility, show from one generation to another changes quite outside statistical error.

Four lines out of the five bred from four pairs are still heterogeneous after 25 generations produced in this way; in the case of the fifth, the correlation coefficient shows that it is not impossible for it to be homogeneous.

Poulson, D. F., and S. J. Counce "Non-mendelizing" phenotypes in D. willistoni.

In the course of extended breeding experiments with Recife-3, Recife-6, and Barbados-3 sex-ratio and reverted sex-ratio lines, flies with "mutant" phenotypes, several of which were comparable to those of known mutants in D. melanogaster, were found. In spite of the fact that more than one such phenotypic variant usually occurred either in the same and/or sister cultures (in pair matings), mutant lines could not be established in subsequent crosses. Only three of eleven phenotypic variants recovered in one breeding experiment proved to be mendelizing.

Among the "non-mendelizing" phenotypes we have encountered are the following:

1. curly wing: perfect mimic of D. melanogaster Cy. Occurs sporadically and regularly in most lines. Flies usually fail to breed, but fertile crosses yield only wild type offspring.

2. cross-veinless: posterior crossvein incomplete or wholly missing with low frequency in some non-SR inbred lines derived from Recife-3 female injected with SR (sex-ratio) ooplasm. Matings of cross-veinless flies yield mostly wild type flies, but the phenotype persists with low frequency in these lines.

3. dumpy-truncate wings: occasional flies with wings that are broad and truncate appeared in the lines showing the cross-veinless effect. Subsequent generations from such flies have given only wild type.

4. slender bristles: all macrochaetes more slender and tapering than wild type, wings tend to be crumpled and rolled. Occurred in offspring of two sisters; in F_1, some "mutant" types were recovered, but yielded only wild type offspring in subsequent generations.

Eye color "mutants":

5. dull: eye color dulled; several flies of both sexes appeared in offspring of two sisters. When mated, produced only wild type in subsequent generations. Similar phenotypes also noted sporadically in other lines.

6. prune: single fertile female with dark purple-brown eyes. Mated to brothers, subsequent generations wild type. Similar phenotype has been recovered in other crosses, but invariably sterile.

7. burgundy: three males in single pair culture with eyes of rich red-brown.

Mated to wild type sisters, subsequent generations wild type.

8. deep orange: one male with eye color similar to that of newly-emerged deep orange mutant of *D. melanogaster*; however, with age, eye color became dull brownish orange. Mated to wild type sisters, subsequent generations wild type.

Poulson, D. F., and B. Sakaguchi Hereditary spirochete infections in Drosophila.

Investigations into the nature of the infectious agent responsible for the maternally transmitted "sex-ratio" condition (SR) in the *willistoni* group of species by ourselves and by C. Malogolowkin in Brazil have made it clear that in three species, *willistoni*, *nebulosa*, and *equinoxialis*, the production of unisexual progenies (SR) is completely correlated with the presence in the hemolymph of numerous minute spirochetes. Furthermore, when SR is transferred from *willistoni* to *melanogaster* it is invariably accompanied by the spirochetes and when SR is lost in *melanogaster* the spirochetes disappear. - Observations on the spirochetes indicate that they are morphologically very similar in the three species, clearly belong to the family Treponemataceae, and have the characteristics associated with the genus Treponema. Typical forms are approximately 0.1 μ in diameter and 4-5 μ in length as seen by phase microscopy. Occasional longer individuals, 8-10 μ in length, appear to represent dividing forms. Under certain conditions the filaments give rise to a minute granular phase (spores ?) from which in tern new spirochetes may later form. Filtration experiments indicate that the frequency of SR infection is not reduced by passage through 0.3 μ filters and that considerable infectious material can pass 100 mμ filters although the period of incubation is increased after filtration. Infectivity of SR hemolymph is completely abolished after admixture with penicillin G. - Although apparently different from a larger spirochete described by Chatton in 1912 from *D. confusa* and named by him *Treponema drosophilae* the rare occurence of giant forms in the blood of very old flies does raise the question of a possible relationship. Since we have demonstrated that SR, and thus spirochetes, may be carried in latent condition for some generations, the whole question of inter-relationships of spirochetes and Drosophila requires thorough investigation. Through the kindness of G. E. Magni we have examined the SR strain of *bifasciata* and thus far have not been able to detect typical spirochetes in the hemolymph. This is in line with the experimental results of Rassmussen who was unable to obtain SR infections by injection of SR hemolymph into normal flies. The SR agent in *bifasciata* appears to be strictly intracellular and whether it is a spirochete remains to be discovered. - The three cases of hereditary treponematosis in Drosophila all derive from regions (Jamaica, Haiti, Puerto Rico) in which the classical treponematoses, syphilis and yaws, are endemic. It is known that flies of the genus Hippelates may serve as mechanical vectors of yaws, although whether these or other insects may carry them transovarially is not known. To what extent the treponemata of Drosophila may prove pathogenic to other forms, including Drosophilists, remains to be examined although it seems a priori rather unlikely. - We hope that Drosophilists will be on the watch for possible cases of spirochetes in various species and strains and shall be grateful for any materials sent to us for study.

Ratty, F. J. Mutation rates at the white and Notch loci of w^{m4} in pre- and postmeiotic germ cells.

A study of mutation rates in the w^{m4} chromosome and normal chromosomes (Lefevre, Ratty and Hanks, 1953) indicated an increased frequency of white and Notch mutations in mature sperm. The present study has attempted to determine whether the mutation rate of these loci is comparable in pre- and postmeiotic *male and female germ cells*. The work reported herein is based upon the examination of 195,000 F_1 of both sexes derived from irradiated males and 172,000 F_1 of both sexes derived from irradiated females. Approximately one half of all progeny were obtained from single irradiated parents to determine the frequency of clusters. No clusters of white, Notch or white-Notch mutations were observed; however, the possibility is not precluded by this experiment because of the low fertility of the tested chromosome beginning 14 days after irradiation. The procedure has been to irradiate the w^{m4} parents with 3000 r. and mate them to y w spl f. The parents remain in the original culture for the first 4 days and are then subcultured every three days for a maximum period of 24 days following irradiation. A comparison of the mutation rates in *mature male and female germ cells indicates an excess* of white mutations in the latter type of cell whereas just the opposite relationship exists for the Notch (non-white) mutants. In the period 7-12 days after irradiation an excess of all types of mutations is found in spermatocytes. However, subsequent to this period, not one white, Notch or white-Notch mutation was found following the examination of over 24,000 F_1 females derived from irradiated males, whereas two Notch and one white-Notch mutants were recovered from an equal number of F_1 females of irradiated w^{m4} females up to 18 days and one white male viable mutant was found after 23 days. Although more data is required in the later periods of this experiment, it would appear that both viable and lethal mutations are either produced more readily in premeiotic female than male germ cells or they are transmitted with greater facility.

(This work was supported by a grant from the National Science Foundation, Contract G6391).

Reddi, O. S., and C. Auerbach Induction of translocations in spermatogonia of Drosophila by CB 1506 (2-chloroethyl methane sulphonate). This substance, as shown by the Fahmys, exercises its major effect on spermatogonia. Adult males were injected with 0.2 µl of a 0.2% solution of CB 1506 and sex-linked recessive lethals and translocations were scored in progeny obtained after 14 days of sexual activity. The results were as follows:

Sex-linked recessive lethals

No. of X-chromosomes scored	No. of lethals	% of lethals
778	256	32.90

Translocations

No. of sons scored	No. of translocations	% of translocations
906	15	1.65
	Control	
550	-	-

Thus, translocations can be produced in spermatogonia by a mutagen that acts strongly at this stage of spermatogenesis, although the frequency in relation to that of sex-linked lethals was considerably below what would be expected from X-rays acting on post-meiotic stages. A peculiar feature was the fact that all 15 translocations involved the Y chromosome, either together with one of the large autosomes or - in one case - with both. This aspect is now under study.

Sandler, L., P. Hart, and B. Nicoletti Phenotypic changes associated with chromosome rearrangements at the y locus. A spontaneous mutant was recovered from a wild-type stock that showed the phenotype of y^N (i.e. wild-type body and yellow bristles). This mutant has been symbolized y^{bl} (yellow bristles). Phenotypically, y^{bl}/y^1 is y^{bl}, y^{bl}/y^+ is y^+, and y^{bl}/y^2 is y^+.

From the cross, $y^{bl}\ w^a/y^2\ w$ ♀♀ X $y^2\ w$ ♂♂, two strange observations were made. First, it was found that crossing over between y^{bl} and w was very much reduced (0.21% as compared with the standard value of 1.5%). Second, two exceptional male progeny were recovered from this cross. One of these was phenotypically $y^1\ w^a$, very aneuploid looking, and died within 24 hours leaving no progeny. The other exceptional male was $y^+\ w^a$, sc-variegated, viable and fertile. When this male was crossed to y f:=/Y females, $y^+\ sc^v\ w^a$ males, y f females, and y^+ f females were recovered in about equal numbers. This latter result, of course, suggested a translocation between the tip of the X including y (which has now become wild-type) with the tip of an autosome such that aneuploidy for the autosomal tip is viable. It is at least interesting that the $y^1\ w^a$ aneuploid recovered from the original cross is also understandable in terms of a similar translocation with the particular male recovered being deficient for the y region.

Salivary gland chromosomes of y^{bl} and the y^+ translocation derivative were examined. These examinations revealed that y^{bl} has salivary bands 5A-5D inserted in reversed sequence as a duplication just distal to the y locus (i.e. just distal to band 1B1); bands 5A-5D are also in their normal position. The y^+ translocation, T(1;2) y^{bl}, is a translocation between the tip of the X and the tip of the left arm of Chromosome II. The breakpoint in the X, however, is slightly proximal to the y^{bl} insertion breakpoint being in bands 1B2-3.

Analyses were performed to specify the translocation breakpoints genetically. A cross of $y/T(1;2)\ y^{bl}/al$ X y/Y, al/al was made to determine whether the al^+ locus had been translocated to the X. The absence of y; al^+ females from this cross indicated that al^+ remained in its original position (0.0 on II L) and therefore the break in II L in $T(1;2)y^{bl}$ is distal to al^+. To determine whether the entire tip of the X had participated in the translocation, a test for the absence of l(1)J1 on the translocation y- X chromosome was made. The test was a cross of $T(1;2)y^{bl}$ males by females carrying Muller-5 and a ring chromosome marked by y^4 f but deficient for l(1)J1. The absence of the female class carrying the proximal piece of $T(1;2)y^{bl}$ and the $l(1)J1^-$ ring indicated that l(1)J1 had been translocated to chromosome II along with the y locus. Finally, a cross was made between y ac sc pn males and females heterozygous for M-5 and $T(1;2)y^{bl}$. The y female F_1 were phenotypically y ac sc^1 indicating that at least ac^+ had been translocated to chromosome II and also the sc locus unless the sc variegated allele now present in $T(1;2)y^{bl}$ is recessive to sc^1. This matter was definitively resolved by showing that the translocated tip of the X covered a sc deficiency. Therefore, genetically $T(1;2)y^{bl}$ is a two-break rearrangement with the break in chromosome II being distal to al^+ and the break in the X being just proximal to sc^+ and resulting in sc variegation.

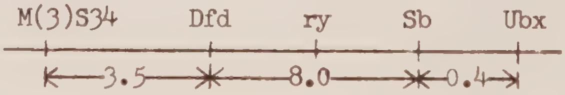
These results, then, show that (1) a phenotypic change at yellow (y^+ to y^{bl}) is associated with a complex, spontaneous, rearrangement of the X and (2) a further phenotypic change at yellow (y^{bl} to y^+) is again associated with a further spontaneous chromosome rearrangement involving approximately the same segment. This suggests the possibility of something akin to the transposable phenotype modifiers of maize with the difference that here, transposition implies chromosome aberrations. This matter is under investigation.

Schalet, A., and A. Chovnick A crossover-selector system for the study of pseudoallelic recombination in D. melanogaster.

The analysis of structural and functional organization of the genetic material in Drosophila is limited by (1) absence of selective sampling procedures in recombination studies, and (2) dearth of known genes with well-defined chemical phenotypes. Due to the efforts of several workers (Hadorn and Schwinck, 1956, Nature 177:940-941; Forrest, Glassman, and Mitchell, 1956, Science 124:725-726; Glassman and Mitchell, 1959, Genetics 44:153-162), biochemical analysis of gene action at the rosy locus (3:51±) has proceeded to a point where it is possible to relate mutations to enzyme alterations. The development of a selective procedure for study of recombination between rosy mutants would make this system a most useful tool for the study of gene structure and function in Drosophila.

A lethal selector system patterned after one devised by Whittinghill (Science 111:377-378) for study of induced crossing over in Drosophila males, has succeeded in resolving two very closely linked rosy mutants (ry^1 and ry^2).

The lethal markers used and their relative map positions based upon preliminary linkage studies follows:



Two series of matings, differing in specific distribution of lethal markers in the female parents, were used in the initial experiments:

Series A

♀♀ $\frac{M(3)S34\quad Dfd\quad ry^1\quad +\quad +}{+\quad +\quad ry^2\quad Sb\quad Ubx}$ x ♂♂ $\frac{+\quad Dfd\quad ry^2\quad Sb\quad +}{M(3)S34\quad +\quad ry^2\quad +\quad Ubx}$

Series B

♀♀ $\frac{M(3)S34\quad Dfd\quad ry^2\quad +\quad +}{+\quad +\quad ry^1\quad Sb\quad Ubx}$ x ♂♂ $\frac{+\quad Dfd\quad ry^2\quad Sb\quad +}{M(3)S34\quad +\quad ry^2\quad +\quad Ubx}$

Due to the homozygous lethality associated with the markers M(3)S34, Dfd, Sb, and Ubx, all offspring of these crosses die, with the exception of the following crossover classes: $\frac{1}{4}$ of the crossovers between M(3)S34 and Dfd, $\frac{1}{4}$ of the crossovers between Sb and Ubx, and $\frac{1}{2}$ of the crossovers between Dfd and Sb. On the basis of preliminary linkage data, these matings should yield approximately 5% surviving zygotes, and these all being crossovers in the region between M(3)S34 and Ubx. Experiments in which eggs were counted, and their subsequent fate noted, confirm the 5% survival figure. Moreover, none of the lethals kill in the egg stage, and most of the death occurs in pupae. In these experiments, since most offspring die, massive matings are made. Collection of large numbers of virgin females is facilitated by incorporation into our base strains of a sterilizer system which permits collection of automatic virgins (see Muller, DIS 23:110).

In these first experiments, eight ry^+ offspring, exhibiting marker recombination, were recovered among 20,235 surviving offspring from Series A crosses. Series B crosses yielded no ry^+ flies among 11,940 surviving offspring. In the Series A crosses, the ry^+ chromosomes recovered were wild type for all markers in the cross. They were found as single offspring from different matings, each carrying a chromosome with male parent markers. Both sexes are represented among the rare ry^+ offspring.

The recombination data of this experiment indicate that ry^1 and ry^2 are separable, with ry^1 to the left of ry^2. The absence of ry^+ flies among Series B offspring confirms the relative position of these mutants, since this crossover class should die in the Series B crosses. Series B crosses should permit survival of the double mutant type. Unfortunately, we have been unable to distinguish exceptional phenotypes which might be double mutants. Estimation of the map distance, ry^1-ry^2, obtained by

$$\frac{2\ (\#\text{ of rosy}^+\text{ recombinants})}{20\ (\text{Total Surviving Offspring})} \times 100 = 0.00393.$$

This investigation was supported by a research grant, C-4440, from the National Cancer Institute of the National Institutes of Health, Public Health Service, and by a research grant, NSF-G6431, from the National Science Foundation.

Schwinck, I. Studies on maroon-like and rosy.

For the genotypes ma-l and ry there had been reported a similar deficiency in xanthine oxidase activity; however, Glassman found a maternal effect correlated with an increase in enzyme activity only for ma-l and $ma\text{-}l^{bz}$. A similarity between ma-l and ry also exists in the aberrant morphology and function of the Malpighian tubes (shorter, irregular formed and puffed up, containing yellow to orange pteridine globules). In ma-l males from y f:= mothers, maternally affected in respect to eye color, the Malpighian tubes were normal; thus indicating a close relation of the aberrant function of the Malpighian tubes and the drosopterin formation of the eyes.

Transplantation of ma-l; cn imaginal eye discs into cn hosts demonstrated a non-autonomous drosopterin formation similar to that reported earlier for rosy (Hadorn and Schwinck, Zeitschr. fur ind. Abstammungs- und Vererbungslehre 87:528, 1956) and $ma\text{-}l^{bz}$ (Glassman, Ursprung, DIS-33). Transplantation of ma-l; cn imaginal eye discs into cn; ry^2 hosts and the reciprocal experiment resulted in an orange eye color in both the implant and the host eyes. Further studies will have to analyze the functional interrelation of ma-l and ry, and their dependence on milieu factors as well as the nature of the ry^+ factor and the maternal factor x of ma-l.

Schwinck, I., and A. Chovnick Eye disc transplantations with garnet pseudoalleles.

In the garnet pseudoallele series the genotypes g^{50e} and g^2 manifest in flies with small amounts of red pigments (drosopterins), in g^w the drosopterins increase to almost the wild type level; however, the heterozygotes g^{50e}/g^w and g^2/g^w are typical garnet phenotypes. Transplantation of v g^{50e} imaginal eye discs into v g^w hosts demonstrated autonomous formation of the small amount of drosopterins. This indicates that no diffusible factor, deriving from the g^w-host cells, acts on drosopterin formation in the g^{50e} implant eye. Transplantation of v; ry^2 imaginal eye discs into v g^{50e} or into v g^w-hosts resulted in non-autonomous drosopterin formation in the rosy implant, the host eyes were not changed in eye color. Thus, the garnet hosts behaved the same way as donors of the $rosy^+$ factor as a number of other genotypes with reduced drosopterins did in earlier experiments (Hadorn und Schwinck, Zeitsch. fur ind. Abstammungs- und Vererbungslehre 87:528, 1956).

(This investigation was supported by a research grant, C-4440, from the National Cancer Institute of the National Institutes of Health, Public Health Service, and by a research grant, NSF-G6431, from the National Science Foundation).

Seto, F. A study of lethal factors in the wild Drosophila populations of Berea, Kentucky.

As part of a study in which second chromosome recessive lethals were collected for a developmental investigation of their lethal effects, the following data were obtained. In a collection of flies from a wild population made in 1959, about 350 second chromosomes were tested with the Muller's Sifter technic for their lethal content. Of these, 110 contained recessive lethal factors which is a lethal frequency of 31±2.4%. A laboratory population was initiated at that time using females and males from the same collection sites and soon after many of the males used in the isolation of the lethal chromosomes were also added to the same population cage. As the population increased in size, additional population cages were started and maintained for a period of about 8 months. At the end of the period, 236 second chromosomes were tested and 55 of these proved to be lethal for a frequency rate of 23±2.6%. A second collection of lethals was made in 1960, from the same collection sites as in the previous year, and out of 345 second chromosomes tested, 129 contained recessive lethals for a frequency of 37±2.6%. To determine the number of independent lethal factors present in each collection group, cross tests of the individual lethals were made by crossing the different Cy/le lines to each other. 57 lethals from the 1959 collection (Wild 1959), 54 lethals from the 1960 collection (Wild 1960) and 54 strains from the laboratory collection (Lab. 1960) were tested. A summary of the results of these tests is given below:

		Wild 1959	Wild 1960	Lab. 1960
"Allelic" matings/total m.		15/1586	15/1431	43/1431
Percentage "allelism"		1%	1%[1]	3%
Frequency of appearance	1	42	30-36[2]	15
	2	3	5-2	8
	3+	2	1	1
	4+	1	2[1]	4
No. of independent lethals		48	38-41	28

1. Since one lethal occurred 7 X and accounted for 21 out of the 36 allelic matings in this group, it was not included in the computation of the allelism frequency.
2. Data in this group are incomplete; further tests are to be made.

The frequency of appearance refers to the number of times a particular lethal factor appeared in each collection group. Further cross tests were also made between Cy/le strains of Wild 1959 with Cy/le of Lab. 1960. The flies of these two populations were of common origin but differed in population conditions and time of collection. The result of the cross tests is a low "allelism" rate of less than .28% (4/1440). An allelism rate of this magnitude has been shown by others to indicate an independence in the origin of the lethals. From the results obtained by others one would expect that most of the lethals in the original population would be eliminated in time and the lethals currently present in the laboratory population would be the product of recent mutations. Although the data are not presented in this research note, a comparison of the frequency distribution of lethal types of the laboratory population with that from wild populations shows a significant difference, whereas the ontogenetic distributions of the Wild 1959 and Wild 1960 lethal series were basically alike.

Although the information presented here is somewhat preliminary, the following tentative conclusions on the observations are made. The characteristics of the wild populations in terms of lethal content appears to be the same from year to year. A population of similar origin but raised in the laboratory in small population cages appears to lose some of its genetic variability. This change is seen in the reduction in the lethal frequency rate and the repeated occurrence of the same lethal genes. A comparison of the ontogenetic distributions further indicate that associated with this reduction in genetic variability, there is a relative decrease in the number of lethals which affect the early stages in development.

Strangio, V. A. A female-superfemale mosaic in *D. melanogaster*. During the course of X-ray experiments utilizing the ring-chromosome X^{c2}, males carrying this closed X are mated to attached-X females homozygous for the sex-linked markers yellow, vermilion, forked and carnation. Superfemales occasionally survive - these are phenotypically wild-type except for characteristic superfemale features - low viability, sterility, scalloped or blistered wings, abnormal appendages, etc.. One particularly unusual fly that appeared was a female-superfemale, left-right mosaic. It seems likely that at the first cleavage, the ring-X was eliminated from a potentially triplo-X daughter cell from which the left side was derived. All the markers except vermilion display more or less complete autonomy. One very striking feature of the mosaic is the sharp line of longitudinal demarcation between left and right pigment patterns in the thoracic region, but a blurring effect similar to the "border zones" described by Hannah (1953) is noticeable in the posterior abdominal tergites.

Taira, T. Is the double recessive Hn^{r-3} ry homozygote a synthesized lethal? ry strain maintained successively in our laboratory had certainly some degree of pupal lethality depending on culturing temperature, as reported by Hadorn and his co-workers. In co-isogenized ry strain after 26 generations, however, such a pupal lethality of the original ry strain is never observed. Therefore, it seems likely that a recessive lethal gene closely linked with ry gene is responsible for such phenomenon. Such ry strain, nevertheless, has maintained a deficiency of xanthine (pterine) dehydrogenase activity as reported by Glassman and Mitchell (1959) and others. As reported by Taira (1960), co-isogenized Hn^{r-3} strain (classical sign; sed) has the lowest activity of pterine reductase of all sepiapterinic strains, but it has the same activity of xanthine (pterine) dehydrogenase as detected in the homogenate from the wild strain.

According to Bridges and Brehme (1944), ry gene is located at 51± on the 3rd chromosome, and as reported by Lewis (1956) Hn^{r-3} gene should be located at 23.0 on the same chromosome because of an allelic gene of Henna-recessive.

When the two strains are crossed with each other, the phenotype and chemotype of F_1 hybrids are similar to those of the wild type strain as expected. The genotypes of F_2 hybrids were determined by the back cross technique of single pair mating. As the results, prospective Hn^{r-3} ry homozygotes in F_2 hybrids were never observed in about 5,000 flies. This suggests that the double recessive Hn^{r-3} ry homozygote might be an artificially synthesized lethal. A low frequency of Hn^{r-3} ry/+ ry flies was checked in F_2 segregating population. The majority of F_2 hybrids were wild type flies (+ +/+ +; Hn^{r-3} +/+ +; + +/+ ry; Hn^{r-3} +/+ ry). The proportions of Hn^{r-3} phenotype (Hn^{r-3} +/Hn^{r-3} +; Hn^{r-3} +/Hn^{r-3} ry) and ry phenotype flies (+ ry/+ ry; Hn^{r-3} ry /+ ry) were about one fourth respectively. Hn^{r-3} ry/+ ry flies, the frequency of which is very low in F_2 segregates, must be checked again in detail.

Takaya, H., S. Kaji, and I. Inouye Hereditary characters of *D. melanogaster*, evocable through changed media. By rearing larvae of the wild-type flies of *D. melanogaster* with the molasses media containing soybean powder, choline chloride or casein hydrolysate, were brought about malformations of varied organs of the adult flies. The organs which were influenced to become irregular, deficient or duplicated were the compound eyes, antennae, wings, legs, or abdomens. Almost all the irregularities of these organs were found to be inherited in the following generations. Further it was shown that these irregularities of organs were susceptible to selection showing gradual increase in the degree and in the frequency of the occurrence.

The frequencies with which these irregular organs were produced were different in different substances employed and also in different concentrations of the same substance. For example, in the wild-type flies of Oregon stock deficiency of the compound eyes was evoked in 7.4% of the operated specimens by using a 5% of casein hydrolysate, in 2.6% by a 2.5% of choline chloride, and in 1.9% by a 20% of soybean powder. Of these substances employed casein hydrolysate was most effective in producing malformed organs of fly.

Thirty-three different stocks of the wild-type flies were investigated. Flies of all these stocks actually produced malformations in response to the changed media. But in several stocks such as Bôchu, Kirishima, Shioya, Canton-S, Oregon and Quicksand frequencies of the malformations were always higher than the other. In regard to the organs malformed difference was also marked in different stocks. In the stocks of Aoshima, Bôchu, Kirishima, Miyazaki, Shioya and Oregon, most of the malformations produced belonged to those of the compound eye, and in Shirahama, Bikini-Atoll, Swedish-b-6 and Urbana-S abnormalities of the wing and in Quicksand and Canton-S deficiencies of the leg were predominant. On the other hand, malformation of the abdominal segment were met with in nearly all the stocks examined with, however, relatively low frequencies. These differences found in different stocks of the wild-type flies can be regarded as the characteristics of the different stocks which may be referrable to different constitution of the genetical system.

Takaya, H., S. Kaji, and I. Inouye
Selection of the malformed abdomen of D. melanogaster evoked through changed medium.

Rearing of larvae with the molasses medium containing casein hydrolysate sometimes resulted in the formation of those adult flies whose abdominal segments were incomplete or lacking. In the wild-type flies of Quicksand and Riverside, such occurrence was met with respectively in 1.56% (30/1921) and 1.08% (31/2860) of the specimens. These flies when inbred by themselves never failed to be reproduced. By the selection carried out by means of sibmating, numbers of these anomalous flies and degrees of the anomaly gradually became increased, and in the F_{10} generation of selection more than 60% of the flies were anomalous in the abdomen.

In every generation of the selection flies with the normal abdomen were always produced. These flies were shown to be able to produce progeny with the malformed abdomen when they were inbred by themselves. Further it was shown that the ability of these normal flies which was weak in the early course of the selection gradually became stronger with advance of the selection.

By means of the back selection the highest degrees of the malformed abdomen gradually became decreased and numbers of the malformed flies also showed gradual decrease to become nearly zero in the F_{10} generation of selection.

In outcrossing of the malformed flies with the wild-type ones of the isogenic Oregon stock, hereditary transmission of the malformed abdomen was always demonstrated. But the malformations of the lowest degrees were reproduced first in the F_2 offspring but failed to appear in the F_1 generation, while those of higher degrees were always reproduced in the F_1 as well as in the F_2 generations. In these crossings numbers of the malformed offspring were not constant but varied greatly. But there was an apparent tendency for flies of higher degrees of malformation to produce more malformed offspring than those of lower degrees. Throughout the present experiment it was found that occurrence of the malformed abdomen was found about 1.5 times more often in the female than in the male flies. All these results except the last-mentioned one are quite in agreement with those that have been obtained in the inheritance of the deficient compound-eyes evoked through changed media (ref. Takaya, H., S. Kaji, and I. Inouye, 1960).

Tobari, Yoshiko, and D. Moriwaki
Relation between adaptive values and karyotype frequencies in D. ananassae.

The present study concerns two different gene arrangements, IIILA and IIILB, owing to a terminal inversion in the left arm of the third chromosome in D. ananassae. When carriers of the two types were mixed together in a definite initial ratio in population cages, they always came to an equilibrium with nearly the same value, 60% A and 40% B, regardless of the initial frequencies, and the results were repeatable. Accordingly the balanced chromosomal polymorphisms are established in the populations. However, the relative adaptive values of three karyotypes regarding the inversion (two homozygotes and one heterozygote) are not necessarily the same though similar in the replicates. That the adaptive values are not always the same may be caused by some differences of internal environments rather than external ones. Here we can postulate two alternatives: one is the genetic constitution of initial members of a population and the other the relative frequency of the arrangements which changes successively in the same population. According to present experiments the latter seems to be more probable, i.e., the relative adaptive value of a karyotype changes corresponding to frequencies which change during the succession.

Toyofuku, Y. Salivary gland chromosomes of D. nigromaculata with special regard to the chromosomal polymorphism.

D. nigromaculata is one of the common species of the quinaria group in Hokkaido.

The salivary gland chromosomes of this species are characterized by five arms consisting of four rod-shaped arms and a dot-like element. For convenience, they are designated as X, A, B, C, D and E.

Investigations on the salivary gland chromosomes of D. nigromaculata in natural populations of Hokkaido have shown that there occur remarkable chromosomal polymorphisms. Twenty-two different kinds of chromosomal aberrations represented by heterozygous inversions were found to occur in eighty-four samples (Table 1). The inversions were found in the X, A, B, C and D arms, while the dot-like E arm showed no aberration so far studied.

Aberrations	X	A	B	C	D
Terminal complex inversion			3(A)		
Subterminal inversion		2(E)		4(E)	
Subterminal complex inversion	1(B)				
Subterminal inversion - long double inversion		2(A + B + C)			
Median inversion		10(F)	2(D)		11(B)
Submedian inversion		1(G)		3(B)	
Long median inversion		1(D)	1(B)		
Double median inversion	1(A + C)		3(B + C)		
Submedian inversion - Subbasal inversion	4(A)	2(G + H)		3(A + C)	1(C)
Subbasal inversion				7(A)	
Long double inversion		1(A + B)			
Long complex inversion				13(B + D)	
Small inversion					8(A)
	6	19	9	30	20

Traut, H. On the identification of radiation induced deletions of the X-chromosome of D. melanogaster.

The method for detecting radiation induced large deletions of the X-chromosome (e.g. Bateman Heredity 9) involves scoring the frequency of phenotypically y^+ daughters of treated y^+ ♂♂ mated to y attached-X ♀♀. Because the appearance of such females may have various causes (see table 1) we bred them for a further generation (F_2) with y^+ ♂♂. Besides F_2 progenies like (1) and (2) we got sometimes others for which (3) seemed to be applicable. As this assumes premeiotically induced "half-translocations" we had investigated, however, only postmeiotical stages the following explanation seems to be more likely: as Muller and Painter (Z.f.V. 62) have pointed out, the proximal heterochromatin of the X functions in disjunction of the X chromosome from the Y chromosome during meiosis. The induced deletions may include part of this heterochromatin and indeed Lindsley and Sandler (Genetics 43) report primary nondisjunction of deleted X-chromosomes in attached-X females. Therefore we suppose the F_2-phenotype combination +♀, y♀, +♂ to be caused by primary nondisjunction indicating a deletion (1): combining the F_2-phenotypes belonging to deleted X (y♀, +♂) with those expected in the case of nondisjunction (+♀, +♂) one gets the F_2 combination +♀, y♀, +♂.

cause	F_2-phenotypes
(1) deletion	y♀, +♂
(2) detachment	+♀, +♂, y♂
(3) X-autosome "half-translocation"	+♀, y♀, +♂
(4) X-Y-"half-translocation"	y♀, +♂

Tsukamoto, M. DDT resistance and production of the metabolite of DDT in D. melanogaster.

Quantitative comparisons on the production of the metabolite of DDT, Kelthane, were colorimetrically carried out after treatment of DDT. From the results obtained, it may be concluded that DDT-resistant and susceptible strains and their F_1-hybrids can metabolize DDT to its ethanolic derivative Kelthane, but the amounts of Kelthane produced in the body are not directly proportional to the resistance levels of the strains tested. Insecticide TDE, dichloroethane derivative of DDT, is also metabolized to its ethanolic derivative, FW-152.

Such a hydroxylating pathway of metabolism has been found to occur in other insect species too, such as in *D. virilis* and *Blattella germanica*, but not in *Musca domestica*, *Bombyx mori* and *Pieris rapae*.

Ursprung, H. Xanthine dehydrogenase in wildtype, white, and brown *D. melanogaster*.

According to recent findings of P. Auf der Maur (this institute, unpublished) the mutant white synthesizes and continuously excretes traces of isoxanthopterin throughout its life. Also this mutant, if fed glutamine and glycine, is able to form considerable amounts of uric acid as an adult. It therefore seemed worthwhile assaying the enzyme xanthine dehydrogenase which in wildtype converts 2-amino-4-hydroxypteridine to isoxanthopterin and xanthine to uric acid (Forrest et al., Science 124:725, 1956). For the assay the method of Glassman and Mitchell (Genetics 44:153, 1959) was slightly modified. Mature larvae, one day and four days old pupae, one day and ten days old adults, respectively, were assayed. Very high enzyme activity was found in wildtype and both w and bw throughout this period, which explains the ability of adult white to form isoxanthopterin and uric acid.

von Borstel, R. C. Postmeiotic nuclear behavior in uninseminated eggs of *D. melanogaster*.

Doane (Science 132:677, 1960) has noted that meiosis goes to completion in uninseminated eggs of the Oregon-R strain of *D. melanogaster*.

We have used a Feulgen whole-mount procedure (von Borstel and Lindsley, Stain Technol. 34:23, 1959) to investigate the postmeiotic degenerative processes of the nuclei of uninseminated eggs from Oregon-R virgins. Eggs are classified in four categories in table 1 by the type of nuclei contained: normal appearing metaphases, "basket metaphases", multipolar spindles, and pycnotic metaphases. Interphase nuclei were never observed.

The normal appearing metaphases vary from one to three in number per egg. The "basket metaphases" are abnormal, enlarged, polyploid metaphases with the chromosomes usually lying randomly over the spindle. The "basket metaphases" varied from one to five per egg in the sample observed, but there was only one each of eggs containing three, four, and five "basket metaphases". The multipolar spindles are either tripolar or tetrapolar and otherwise closely resemble the "basket metaphases". Occasionally an egg contains both a multipolar spindle and a "basket metaphase"; these eggs were categorized in the multipolar spindle column only in table 1. Though the degenerating nuclei in the column headed "pycnotic metaphases" could often be identified as "basket metaphases", they were not so classified. During the degenerative period, the "basket metaphase" contracts in size and the chromosomes within each spindle round up and fuse. By the sixth to seventh hour after laying, the nucleus consists of one to a few pycnotic spheres in a small area of cytoplasm and there are one to four such areas per egg.

Table 1.
Different types of nuclei found in uninseminated eggs of different ages.

Age in hours after laying	Eggs containing: normal appearing metaphases	"basket metaphases"	multipolar spindles	pycnotic metaphases
2-3	4	44		
3 1/4-4 1/2	1	6	4	1
4 3/4-5 1/4	1	11	4	7
6 1/4-7 1/4				16

Whittington, W. J., and W. E. Peat The behaviour of the Xasta (Xa) mutant of D. melanogaster in populations.

Using small plastic beakers as population cages it has been shown that the Xasta mutant maintains itself despite its lethality when homozygous. The entire population of each cage was counted at frequent intervals and the flies were afterwards replaced in the cages. The method has the advantage of accuracy and speed through the elimination of sampling but the populations are more subject to drift.

Xasta was shown to segregate equal numbers of Xasta and normal gametes in backcrosses, but there was a marked deficiency of normal flies when heterozygotes were crossed. This was found to be due to an accumulation of lethal genes on the normal chromosomes and to greater vigour of the heterozygotes under crowded conditions. Xasta contains a translocation and two inversions, which can be obtained separately in other stocks. Populations were therefore followed containing these inversions and it was found that they were maintained. When the Xasta stock was made homozygous for the inversions Xasta itself disappeared from the population. Evidently the recombination now possible disrupted the favourable gene combinations in the inversions.

Since the two inversions involved occur naturally it seems likely that the irradiation treatment that produced the Xasta mutation superimposed a translocation on the two inversions and the mutant phenotype was thus pre-conditioned to survive.

Yamada, Y. Doubling dose and radiation-induced mutation rate for chaeta characters in D. melanogaster.

The spontaneous and radiation induced mutation rates of polygenic characters with continuous variability are measured by the amounts of variance increment per generation and per unit dose of irradiation, respectively. The rate expressed in terms of variance increment, however, cannot be compared with that in major genes, since the units of measure are entirely different, i.e., the variance in the former, and relative frequency of occurrence in the latter. Despite this, the doubling dose expressed as a ratio of spontaneous and induced variance increase rates provides a common basis for comparing mutability due to radiation, relative to spontaneous mutation, between polygenes and major genes. If the distribution of mutations induced by irradiation is the same as that of spontaneous ones, a doubling dose expressed in terms of variance would indicate as an expectation that induced mutations take place as frequently as the spontaneous mutations by this dose; consequently it has the same meaning as the doubling dose in major genes.

A number of experiments have been conducted in our laboratory in order to estimate doubling doses for bristle number on the 4th and 5th abdominal sternites and that of right and left sternopleura. Based on the genetic variance increase rate per unit dose of radiation between homozygous lines extracted from a single irradiated genome, our estimates of induced mutational variance increase rates were 8.7×10^{-5} and 3.5×10^{-5} per rad, respectively for abdominals and sternopleurals, converted under random mating basis. Assuming the variance increase rate of 0.005 and 0.001 units due to spontaneous mutations for the above two characters respectively, we obtained the doubling doses of 58r and 29r respectively. Other estimates based on the genetic variance increase between heterozygous lines extracted from a single irradiated genome were 38.7×10^{-5} and 13.9×10^{-5}, corrected to the random mating basis. The doubling doses were 13r and 7r. These estimates agree fairly well with the doubling dose obtained for the visible or lethal genes in Drosophila and other animals. However, this does not mean the mutability in polygenes is of the same level of that in major genes.

In order to compare mutabilities in terms of probability occurrence for these two kinds of genes, we should know the number of loci and the average genic effect of polygenes involving the character in question, because the rate should be expressed in terms of relative occurrence per locus per unit dose of radiation. If we could assume 100 loci with the average genic effect of 0.32 for the character of abdominal bristle number, and further Poisson distributions of equal number of positive and negative mutations with the same genic effect, the above rates of variance increased by radiation would be expressed in terms of mutational occurrence per locus per rad to 8.7×10^{-6} for the first test with homozygotes and 4.1×10^{-5} for the second test with heterozygotes. These are very much higher than those in visible genes. If the number of loci involving the character were more than 100, providing the same amount of genic effect, the rate should be less than the above. If a hypothesis of isoalleles at each locus with various genic effects were employed, the mutation rate in polygenes should be higher than that of major genes. Because only one-directional mutations can be detected by any specific-loci-method for measuring mutation rate, even though there are a number of alleles with different characteristics or genic effect at each locus in question.

Yanders, A. F., and J. P. Perras Sperm length in four Drosophila species.

Published descriptions of the species of Drosophila indicate that the testes in the male and the ventral seminal receptacle in the female are quite similar in length within a species. This is to be expected if the testis length is an indication of sperm length, and if sperm are stored longitudinally in the ventral receptacle of an inseminated female. The variation in the length of these organs from species to species, however, can be quite large.

To determine the relationship between length of testes, ventral receptacle, and sperm, we have attempted to measure the length of sperm from selected species whose published descriptions indicate that the lengths of the testes and seminal receptacle are relatively short (e.g., pseudoobscura, persimilis), intermediate (e.g., melanogaster) or long (e.g., hydei, gibberosa, repleta). The problem of consistently obtaining unbroken, isolated sperm, especially in species of the "long" category, has not yet been solved (we have been able to obtain only one measurable sperm from the species in the "long" group), but our preliminary data indicate that the relationship between length of sperm, testis, and ventral receptacle is as anticipated.

To obtain sperm for measurement, the seminal vesicle of a mature male is ruptured in saline, and a few sperm from the mass are transferred to a clean slide, treated with acetic acid-formalin fixative, air dried, and washed with alcohol to remove crystals. Single, isolated sperm are traced with the aid of a camera lucida. The length of the tracing is measured by coating the completed drawing with rubber cement and applying a fine black thread to the sperm outline. The thread can then be removed for measurement, and is kept as a permanent record of the length. Mean lengths and standard errors for four species are as follows:

Species with relatively short testes and ventral receptacle:		
D. persimilis (Rochester	16 sperm	0.279 ± 0.003 mm
D. pseudoobscura (Rochester)	36 sperm	0.295 ± 0.009 mm
D. pseudoobscura (Yale)	13 sperm	0.304 ± 0.003 mm
Species with testes and ventral receptacle of intermediate length:		
D. melanogaster (Oregon R-Chicago)	13 sperm	1.751 ± 0.025 mm
Species with relatively long testes and ventral receptacle:		
D. hydei (Yale)	1 sperm	6.640 mm

The measurements of melanogaster sperm agree well with those of Cooper (in Biology of Drosophila, 1950: 5 sperm; 1.76 ± 0.04 mm), but our figures for pseudoobscura differ slightly from the length reported by Dobzhansky (as cited by Cooper: 0.4-0.5 mm). It seems remarkable that the single measurable sperm so far obtained from hydei is over 3 times the mean length in melanogaster, and nearly 24 times the mean length in persimilis.

Cooper has described the melanogaster spermatozoon as "a most impressive gamete". We suggest that the hydei spermatozoon is even more impressive, and again raise the question as to the possible utility of such unusually long gametes. We hope (see "Materials Requested") to extend our data to include other species, as well as various strains within a species, in order to determine whether sperm length may have a definite taxonomic relationship or is merely a fascinating topic for dinner-table conversation.

Zurcher, C. A factor for dark abdomen present in a wild strain of D. melanogaster.

In a stock of D. melanogaster from Uganda a factor for darker body color was detected and described in an earlier publication (Zurcher, Arch. Jul. Klaus Stift 33:75, 1958). This factor was traced to the third chromosome. Recombination experiments with the genes, st, cu, ca showed that the new mutant gene is close to or at the e-locus. In a standard test for allelism no recombination between it and e could be observed among 19,050 individuals, so that the two factors are probably alleles. It is proposed to name the new mutant ebony-Uganda, e^{ug}. Chromatograms revealed that e^{ug}/e^{ug} genotypes contain approximately the same amount of fluorescent substances as e^{ug}/e genotypes, both genotypes, however, having more such substances than either +/+ or e/e.

TECHNICAL NOTES

Barker, J. S. F. Yet another population cage.

A population cage essentially similar in design to that described by Wright and Dobzhansky (1946), but differing in one feature, has been used successfully in this laboratory. The cage is constructed of 3/8" bonded plywood, dimensions being 15" x 15" x 8". One side consists of a wire mesh screen and the side opposite this has a 5" diameter hole, to which is attached a 15" long nylon gauze sleeve of the same diameter. This sleeve is tied off with cotton tape. The glass lid is held in grooves on two opposite sides of the cage and held on with adhesive tape. If it is required to replace a glass lid that has become too dirty, the adhesive tape is removed, a new lid slid on as the old one is removed, and the new one taped in position.

Sixteen depressions, each 2" diameter, are cut in the floor of the cage. The medium jars (2 oz. pomade jars, 2 1/2" high x 1 5/8" diameter) stand in these depressions. These jars are placed in and removed from the cage through the nylon sleeve. It has been found convenient to use a cycle of 15 medium jars, 5 per week, one each day Monday to Friday. A jar thus remains in the cage for 21 days. Normally 20-25 ml. of a dead yeast fortified medium is used in each medium jar. Under these conditions, a population of D. melanogaster (Oregon-R-C) averages about 4000 to 5000 adults. The sixteenth jar position in the cage is used if it is required to collect egg samples from the cage population.

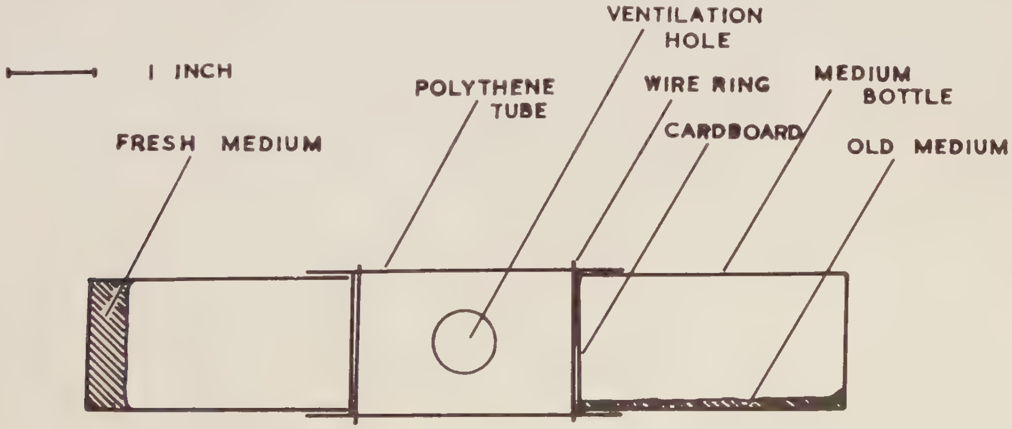
The major advantage of this design is that it allows easy and rapid collection of adults from the cage using an aspirator connected to a small vacuum pump. This is convenient also when it is required to transfer the population to a clean cage as all the adults can be collected in ten minutes or so, much of this time being spent in catching the last few individuals.

Barker, J. S. F. An adaptation of the population bottle of Reed and Reed (1948).

In studies of competition between D. melanogaster and D. simulans in this laboratory, the population bottle has been found to be particularly useful. As pointed out by other workers who have used this technique, the total adult population can be censused conveniently and a large number of populations can be handled simultaneously.

To maintain a population of lower total numbers, but with reduced fluctuations in number, the following adaptation of Reed and Reed's technique has been used.

The population bottle is shown diagrammatically in the figure. It consists of two cylindrical glass medium bottles, each 3" long and 1 9/16" outside diameter, joined by a 3 3/4" long cylinder of polythene tube (40 mm diameter), which fits snugly onto the glass bottles. A ventilation hole (3/4" diameter) is punched from the polythene tube and covered with nylon gauze. The population bottles are stored horizontally in wire trays (9 per tray), with the ventilation hole to one side and the bottle containing the freshest medium towards the light source in the constant temperature room. The medium, when worked by larvae, rapidly becomes semi-fluid and flows down the side of the bottle. To prevent it from flowing out into the polythene



tube, cardboard circles are placed at the mouth of the bottle and held in place by a wire ring. The cardboards are 40 mm wads, plastic finish on one side (these are normally used in the lids of screw-top jars). The plasticised side is placed towards the interior of the medium bottle. Movement of adult flies between the media bottles and the central air space is allowed by means of three 1/4" diameter holes punched in the cardboard.

At the initiation of each population, one medium bottle contains medium, the other being placed on empty. Two weeks after initiation, the population is etherised by holding an ether pad over the ventilation hole. As soon as the flies appear groggy, they are removed and censused. All pupae and larvae are carefully removed from the cardboards and polythene tube, larvae being placed into the medium bottle and pupae into a clean polythene tube, together with the etherised flies. New cardboards are attached and the empty medium bottle replaced by one containing fresh medium. Ten mls. of medium are used regularly in each medium bottle. This censusing is repeated every two weeks, and at each census, the older medium bottle is replaced. A medium bottle thus remains on the population bottle for 4 weeks.

The size of the populations maintained by this technique may be of interest. Twelve populations each of *D. melanogaster* (Oregon-R-C) and of *D. simulans* (vermilion) have been maintained with this fortnightly censusing for 22 weeks on a dead yeast fortified medium. The mean numbers of adults in the populations at each census are given in the table.

	No. of adults censused					
	D. melanogaster (Oregon-R-C)			*D. simulans* (vermilion)		
Weeks after Initiation	No. of Populations	Mean	Standard Error	No. of Populations	Mean	Standard Error
2	12	127.4	8.9	12	174.8	9.8
4	12	316.3	21.1	12	267.1	29.9
6	12	183.3	15.8	12	122.6	23.3
8	12	269.3	28.8	11*	152.7	22.8
10	12	324.8	32.8	11	147.8	20.4
12	12	397.3	47.4	11	201.1	36.8
14	12	273.2	35.8	11	91.6	23.4
16	12	278.8	32.6	11	191.3	38.1
18	12	211.9	21.2	11	115.3	47.5
20	12	350.7	45.7	11	196.0	41.8
22	12	155.0	16.9	11	217.7	47.8

*One *D. simulans* v population bottle had no adults or immature stages present at the fourth census. This is the only population in some 300 studied that has become extinct, though it should be mentioned that many of these populations were maintained for only 3 censuses.

Farnsworth, M. W. Handling Drosophila eggs during fixation.

Fixation of eggs in slowly penetrating reagents as osmic acid usually requires that the eggs be pricked in the fixing medium. Osmic acid is quite toxic and pricking the egg in this medium beneath the microscope involves the inhalation of fumes by the operator. We have avoided this hazard by the following procedure: eggs are collected and may be washed free of adhering food if desired, but are not dechorionated. Scotch tape, sticky side up, is then fastened to a glass slide. The eggs are transferred to the Scotch tape with a medicine dropper and the water is drawn off by filter paper or fine pipette. The chorion of the egg sticks fast to the tape and many eggs can be placed on the same piece of tape and handled at the same time. The section of tape is cut off the slide and placed in a vial of fixative. After 20 minutes to one-half hour, the sections of tape can be removed from the fixative by forceps, placed in water, pricked, and returned to the fixative. The cellophane backing of the tape usually dissolves loose from the crepy adhesive surface during the course of fixation. The eggs can easily be freed from the adhesive at any time by tearing the chorion with a fine needle and allowing the egg to float free.

Iyengar, Shanta V. A method for localized treatment of the polar cap of Drosophila with chemicals.

A simple small apparatus has been devised and constructed for localized treatment of the polar cap of D. melanogaster with chemical solutions to induce mutations. The polar cap stage of the embryo is characterized by the location of the cells of the early germ track at the posterior end of the egg in the form of a cap.

The apparatus consists of a horizontal piece of soft polyethylene translucent tubing about 2 1/2 inches long and 3/8 inches wide attached at each end to glass tubes bent up at right angles. The vertical ends of the glass tubes are about 1 1/2 inches long. The upper surface of the polyethylene tubing is pierced by a beading needle under a dissection microscope thus controlling the size of the pore. About 120-150 pores are made of about 130-145U diameter, the diameter of the eggs being 150U on the average, although they tend to vary somewhat in size. Since the polyethylene tubing is transparent it can be easily observed if air bubbles are present and by tilting the whole apparatus in one or the other direction they can be expelled. The level of fluid in the tube during treatment should be just enough to fill up to the horizontal level of the glass tubing as larger amounts tend to allow the chemical to run out through the numerous pores. The atmospheric pressure and gravity help to keep the fluid down to maintain its level. The whole apparatus is fixed to a glass plate by Scotch tape holding down the polyethylene tube at either end. To ensure against drying a long glass butter dish lined with cellucotton moistened with water could be used to cover the apparatus. The eggs are placed as follows: with the help of a fine dissecting needle dechorionated eggs are picked up from a rectangular piece of black filter paper and placed polar cap downwards in the pores in a sequence and removed in the same sequence so as to allow for all of the eggs the same time for treatment.

The advantages of this technique are: 1) that it allows highly localized action of the chemical with a minimum of capillary action, thus the lateral part of the embryo is virtually unaffected by the chemical; 2) the pores are just the size for the eggs so that only the polar caps are directly exposed to the chemical even if volatile (formaldehyde); 3) the same apparatus could be used for a chemical all through the experiments, thus ensuring maximum uniformity in treatment; 4) for chemicals that are non-volatile even the same fluid could be used many times.

From the practical point of view the apparatus is most easy to handle - especially in the case of carcinogenic substances - all chemical solutions merely being pipetted into or out of the tube by means of a long-drawn-out pipette.

Other methods of treatment of the polar cap have been baths or submersions for whole eggs or dechorionated eggs. Also a rigid plastic tube of the above dimension with one or two channels just wide enough to place the eggs in a row could be substituted for the soft polyethylene; however, the above method with pores seems to be best as the eggs tend to lie flat if not supported, thus exposing other parts than the polar cap to the chemical.

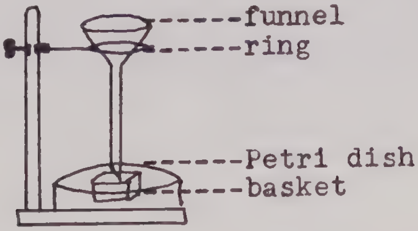
Kaplan, W. D. Snail mantle tissue as a matrix for processing Drosophila eggs.

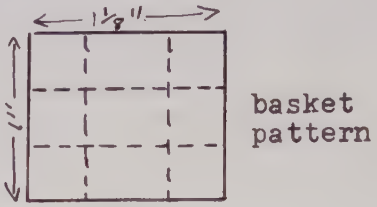
A method for the orderly handling of large numbers of Drosophila eggs for cytological study has been developed using snail mantle tissue as a matrix. The mantle tissue of Helix aspersa may be cut into 5 mm squares and eggs of D. melanogaster are then arranged in parallel rows on the external surface. The mantle secretes a large amount of mucus which, when hardened, acts as a cement to hold the eggs in place in the desired manner of orientation. The mucus requires from 5 to 30 minutes to dry depending upon the thickness of the mantle and the viscosity of the mucus. When dried the eggs will be held in place and the square of tissue may then be carried through the various steps of fixation, dehydration, and embedding without any of the technical difficulties usually attendant upon handling small objects.

Lazarus, H. D., G. L. Kelly, and R. Sederoff. Improved laying-dishes and a refined technique for collecting and washing eggs.

Materials: A ring stand with a ring, a glass funnel, a 1.0 liter pyrex Erlenmeyer flask, two squeeze bottles, one containing distilled water and the other containing distilled water with just enough detergent added to break the surface tension, a quantity of #100-mesh brass strainer cloth, and as many Petri dishes (2-inch and 4-inch diameters) and Stender dishes (2-inch diameters) as are needed for a particular experiment.

Set up the stationary apparatus as shown in the first figure below. The egg baskets are to be made of the brass strainer cloth and cut about the same size as shown in the second figure below. Fold the cloth along the dotted lines shown in the diagram and tuck in the corners so that a little basket is formed.





Laying-dish preparation: 1) Mix 5.0 grams of corn meal, 8.0 grams of agar, and 250.0 milliliters of water in the Erlenmeyer flask, to make enough medium for about 20 laying-dishes. 2) Autoclave this mixture at 250.0°F under 18 pounds pressure for at least 15 minutes to dissolve the ingredients into a homogeneous medium. 3) Pour a 1/4 inch layer of this medium into 2-inch diameter Petri dishes. 4) Allow the medium to cool at room temperature. 5) Gently rinse the surface of the medium with tap water in order to remove any loose pieces of agar which could introduce nutrients into the egg baskets during the egg washing procedure. 6) Let the surface of the medium evaporate to dryness (about ten minutes). 7) Make a thin paste of active dry yeast and water. 8) Spread the yeast paste onto the surface of the medium with one clean finger or a small brush. Leave a 1/4 inch margin between the rim of the laying-dish and the yeast. 9) Allow the surface of the yeast to become dry (about ten minutes). 10) Put the flies to lay immediately. Half pint milk bottles work well as parental bottles with these laying-dishes.

Egg collection and washing: 1) Remove the parental flies from the laying-dishes when enough eggs have been laid for your purposes. 2) With a tweezers, pick off the dead flies, larvae, pupae, or other gross debris, which may have fallen onto the laying-dishes from the parental bottles. 3) Wash the yeast and the eggs through the funnel into the baskets with a fine stream of detergent solution from a squeeze bottle. 4) The eggs should remain in the baskets, and the yeast and other soluble impurities will have passed through the baskets into the Petri dishes. 5) Alternately direct streams of distilled water and detergent solution into the baskets to rinse the eggs and remove any other remaining contaminants. 6) Examine the baskets and eggs under a 10-X dissection microscope and, by using a tweezers or an eye dropper, remove any pieces of medium or other particles which may have been washed into the baskets. 7) Put the egg baskets into Stender dishes containing a 70 per cent ethyl alcohol solution. A 45 minute exposure will render the eggs aseptic. 8) Absorb excess alcohol from the baskets onto a paper towel previously sterilized within a 4" diameter Petri dish. 9) Put the baskets onto prepared aseptic non-nutrient agar Petri dishes and allow the larvae to hatch (14 to 18 hours). 10) The larvae can be collected off of the surface of the agar and inoculated onto a defined medium, aseptically, by means of a heat-sterilized microspatula or a bacterial inoculating needle. Be sure to cool the inoculating tool before transferring the larvae.

Conclusions: 1) About 100 "ripe" females may lay 50 to 100 eggs in 2 to 4 hours. 2) The time involved in egg collecting and washing has been reduced to nearly 25 per cent of the time required by older methods, i.e., about one hour of time is required for this complete procedure for 12 baskets, 45 minutes of which is taken by the alcohol sterilization procedure. 3) The eggs reach the baskets relatively free from impurities, due to the hard surface of the new laying-dish medium. 4) Any flakes of yeast which reach the baskets are easily dissolved with a few squirts of detergent solution. 5) The number of intact eggs obtained in this manner exceeds the number obtained by other methods, by as much as 50 per cent, since nearly 100 per cent of the eggs on the laying-dishes are washed into the baskets undamaged. 6) Apparently the only limiting factor, to the number of eggs collected, is the laying ability of the adult females. 7) Approximately 75 per cent of the eggs laid hatch on the aseptic non-nutrient agar Petri dishes.

Lewis, E. B. A new standard food medium.

The medium described below has been in continuous use at CIT for the last five years. It gives greater yields of flies and shorter emergence times than do media incorporating only propionic acid or tegosept as mold inhibitors. The food contains agar, corn meal, dried brewer's yeast, dextrose and sucrose. A mixture of phosphoric and propionic acids acts as the mold preventative. The medium is seeded after cooling with "Y-2 Original", a live yeast strain isolated by R. P. Wagner (Univ. of Tex. Publ. No. 4445:104-128). This yeast was found by Wagner to tolerate amounts of propionic acid which are quite inhibitory to molds. Since the yeast forms a relatively dry and non-sticky layer as it grows over the surface of the food, flies do not get trapped in it as so frequently happens with conventional live yeasts. Wagner's yeast has been maintained in pure culture and used with a propionic acid media in this and a number of other laboratories for many years. The new feature is the incorporation of phosphoric acid as a means of lowering the pH of the food. This makes it possible to reduce the amount of propionic acid needed to act as an effective mold inhibitor, since only the undissociated form of propionic acid is fungicidal (see discussion by S. Mittler DIS 21:90). The food recipe and the method of maintaining and using Wagner's yeast are as follows:

Food Formula

Ingredients	Composition	Amounts for 300-bottle batch
Water	81.8%	13,000 ml (for dissolving agar) 4,000 ml (for wetting other ingredients)
*Agar	*0.45%	*93 gms.
Dextrose	5.0%	1,033 gms.
Sucrose	2.5%	517 gms.
*Corn Meal	*8.3%	*1,716 gms.
Dried Yeast	1.5%	310 gms.
Phosphoric Acid	0.06%	200 ml of ACID MIX "A" (see below)
Propionic Acid	0.4%	

*Since the amounts of agar and corn meal required to give the correct consistency vary not only with brand but also with different climatic conditions, some adjustment in the amounts of these substances may be necessary before the proper consistency is found.

Preparation of Acid Mix "A". A stock solution containing the appropriate mixture of phosphoric and propionic acids is prepared as follows: to 836 ml of propionic acid add distilled water to bring volume to one liter. To 83 ml of phosphoric acid (H_3PO_4 85%) add distilled water to bring the volume to one liter. Combine the two diluted acid solutions to obtain approximately two liters of ACID MIX "A". This mixture has a pH of between 0.8 and 0.9. To prepare the 300-bottle batch, for example, measure out exactly 200 ml of ACID MIX "A" and add to food after the food is cooked and stir in well. After cooling and setting, the pH of the food ranges between 4.2 and 4.3. The pH of the food is measured by inserting the electrodes of the pH meter into a beaker containing a mash of food that has been removed from one of the culture bottles after the agar has firmly set.

Food in this laboratory is ordinarily prepared only in 150, 300 or 450 bottle batches. A month's supply of ACID MIX "A" is made up at a time and colored with a green food dye. The dye serves later as a check to be sure that it has been added to the food. A set of weights for each of the ingredients is prepared by pouring lead shot into polyethylene bottles until the combined weight is the exact weight of the ingredient for the batch in question. The bottle is then sealed and conspicuously labled by painting on it the name of the ingredient and the batch size. Different colors are used for the different batches to avoid mix-ups. In this way time is saved in weighing out the ingredients and the danger of errors in weighing is reduced.

Culture medium for strain Y-2 original of R. P. Wagner. Procedure: To 500 cc of hot tap water add 5 gms of Bacto-peptone, 10 gms of dextrose, 1 gm of K_2HPO_4 and 0.25 gm of $MgSO_4$. Stir until dissolved. Then remove 100 cc of this mixture to another beaker and add to it 2 gms of powdered agar. Boil this latter batch until the agar is dissolved, stirring constantly over a low flame. From this pour a few tubes.

From the remaining 400 cc of the original mixture (not containing agar) pour about 30 test tubes - about 10 cc per tube.

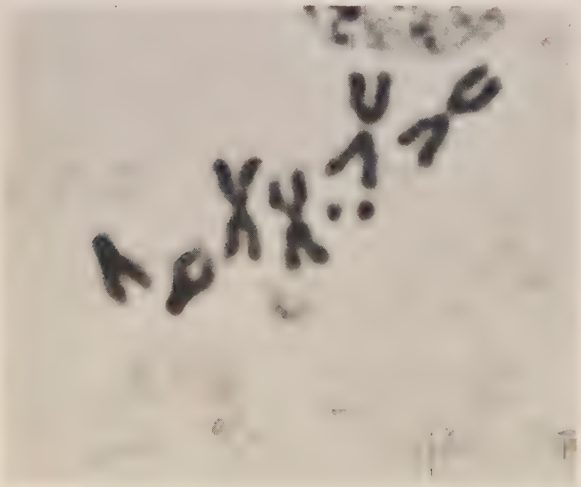
Stopper all tubes tightly with cotton plugs; autoclave at 15 pounds pressure for 20 minutes. When the tubes are removed slant the agar tubes. After cooling inoculate agar and liquid tubes, using standard bacteriological sterile techniques, from an agar slant culture of this yeast. After one day's growth at room temperature it is best to store the yeast tubes in the cold (say 10-20°C).

Inoculation of culture medium with Wagner's yeast. One of the test tubes containing a liquid culture of the yeast is diluted with an equal volume of tap water. The mixture is poured into an atomizer and very lightly sprayed over unstoppered bottles containing freshly prepared fly food medium. (Before spraying with live yeast, the fly food is allowed to cool. During the cooling period the bottles are left unstoppered but are covered with thick layers of gauze. Circulating fans are used to hasten cooling and evaporation of water of condensation.) A few sprays from the atomizer suffice for a 300-bottle batch. The bottles are then papered and stoppered immediately.

Lewis, E. B., and Linda Smith Riles A new method of preparing larval ganglion chromosomes.

A modification of methods currently in use for mammalian somatic chromosomes makes it possible to prepare larval ganglia with large numbers of mitotic figures having well spread and flattened chromosomes. Briefly, the method consists of feeding larvae on colcemide, dissection and treatment of the ganglia in a hypotonic citrate solution, and staining of the ganglia in two stages with orcein.

The solutions required are as follows: Solution (A) is prepared by dissolving 0.1 gm of crystalline colcemide in 100 cc of double distilled water. It is stored in the refrigerator. (Colcemide is a colchicine derivative produced by CIBA Pharmaceutical Products, Inc., Summit, N.J.). Solution (B) is a thick suspension of dried brewer's yeast (20%) and sucrose (5%) prepared in distilled water. It is kept loosely stoppered in the refrigerator and made up fresh from time to time. Solution (C) is a hypotonic sodium citrate solution made by dissolving 1 gm of $Na_3C_6H_5O_7 \cdot 2H_2O$ in 100 cc of double distilled water. Solution (D) is a fixative consisting of equal parts of 45% acetic acid and 95% ethyl alcohol. Solution (E) is a stain composed of 4% synthetic orcein (Gurr's) dissolved in 45% acetic acid. Solution (F) is a stain composed of 2% synthetic orcein (Gurr's) dissolved in a mixture of equal parts of glacial acetic acid and lactic acid (85%). (Filtering of solutions E and F is not necessary and they should be prepared without heating. Natural orcein although excellent for salivary gland chromosomes is unsatisfactory for staining ganglion chromosomes.)



The procedure is as follows: Third instar larvae that have left the culture medium but are still actively crawling on the paper toweling are collected, washed quickly in distilled water, and placed in the center of a two-inch square of Kleenex which is then folded up in such a way as to prevent the larvae from escaping readily. The resulting packet is placed in a small glass dish and saturated with a yeast-sugar 0.05% colcemide solution made up by mixing equal parts of solutions A and B. About 2 or 3 cc should suffice to saturate the Kleenex. The dish is then covered to prevent the larvae from escaping. After a minimum of two hours and a maximum of three hours of feeding on the colcemide, larvae are removed, rinsed and dissected in drops of solution C that have been placed on a siliconed slide. (Larvae which have escaped from the paper and crawled up on the sides of the vessel should be discarded.) The ganglia (dorsal and ventral) are dissected out as a unit, freed of adhering imaginal discs and transferred to a fresh drop of solution C on the same slide. After 10 minutes in solution C they are transferred to a drop of solution D for approximately 30 seconds; they are then transferred to a small drop of solution E which has been put in the center of a siliconed coverslip; after staining 10 minutes in solution E an equal sized or larger drop of solution F is placed next to the first drop and the two drops run together with the tip of a dissecting needle. After 10 minutes a clean slide (not siliconed) is inverted over the coverslip and, as soon as the stain makes contact with the slide, the slide is quickly lifted up, turned over, covered with a Kim-wipe and squashed by thumb pressure on the coverslip. The slide can then be examined as a temporary mount or made permanent by leaving on a block of dry ice for twenty minutes, flicking off the coverslip with a razor blade, immersing for 5 minutes in 95% alcohol, one minute in absolute alcohol followed either by xylol and permount or by going directly to euparal or the Zeiss L15 mounting medium. The reason for the two stage staining technique is to avoid the bursting of nuclei which occurs with acetic-lactic orcein alone or the failure of nuclei to flatten if only 45% acetic orcein is used.

Because of the hypotonic treatment the staining tends to be somewhat weak so that it is desirable to examine preparations with phase contrast. Poorly stained preparations have been satisfactorily restained by the Feulgen method.

With these procedures each ganglion yields many metaphase plates. The chromosomes take on the typical appearance seen in mammalian chromosomes subjected to colchicine and hypotonic treatments. There tends however to be more separation of sister chromatids in Drosophila so that the two pairs of large autosomes appear as four X-shaped bodies. The two X chromosomes of the female appear as a pair of V-shaped chromosomes. The dot chromosomes only occasionally show evidence of a split. Most remarkable is the Y chromosome which appears as a rod at metaphase with the two sister chromatids very closely apposed. Often the centromeric regions of X, Y, or the major autosomes are clearly in evidence as non-staining segments. The same technique applied to imaginal discs yields abundant mitotic figures but with somewhat smaller chromosomes. The method is particularly valuable for examining ring chromosomes.

Mickey, George H. New caps for milk bottles.

In place of the cardboard caps for standard milk bottles, the new polystyrene caps work for Drosophila cultures. The cap fits over the mouth of the bottle and has a small tab on the side which makes it easy to handle. Of course, holes must be punched in the caps since they are air tight. (Manufactured by Owens-Illinois, 70 Sewell Street, Glassboro, N.J.)

Schuellein, R. J. A modification of the salivary-gland chromosome smear technique.

The following smear technique has been successfully employed in student laboratory studies with consistently good results. Essentially it follows the J. Schultz method as reported by B. Nicoletti in DIS 33:181-82, 1959. The modification has a two fold advantage. It eliminates the use of dry ice and substitutes the dehydrating agent dioxane in place of alcohol. Dioxane does not wash out the orcein stain as readily as alcohol. The procedure is as follows:

1) A number of clean siliconized slides are ringed with white vaseline. The ring is made by dipping the open mouth of a shell vial into a jar of vaseline and transferring a thin circle of vaseline onto the slide with a rotating movement of the vial.

2) A drop of lactic acid orcein is placed in the middle of the vaseline ring.

3) A coverslip previously coated with a thin film of Mayer's albumen is flamed and held in readiness.

4) The salivary gland is removed according to Schultz's technique and added to the stain.

5) The usual staining time and squash technique is then followed.

6) When a permanent mount is desired the temporary mount is immersed in a petri-dish containing dioxane.

7) A Novy cover glass forceps (Will Lab. Supply Co.) is used to pry the coverslip from the slide. The Novy forceps may be readily substituted for by snipping the end of a teasing needle and honing the blunt end into a long tapering chisel-shaped probe.

8) The coverslip, to which the chromosome preparation is attached, is immersed in the following washes: a) Dioxane - 30 seconds; b) 50% dioxane-50% Xylol - one minute; c) Xylol - one to two minutes, then mount in a suitable medium.

Stalker, H. D. Improvements in cup-trapping techniques. Collecting wild Drosophila with the bait held in cups, while sometimes the most convenient method, may have serious drawbacks such as ultimate leaking of the cups, loss of bait in heavy rains, and the length of time required to set and take down a large number of cups. The following methods eliminate some of these disadvantages. The bait cups are 8 oz. soft plastic tumblers, with a flare at the lip, and colored black on the outside with spray enamel. The inside is roughened with sandpaper to permit the flies to move down to the bait quickly. Rain caps are prepared from paper picnic plates, folded into a roof-shape, with the outer surface sprayed black. A small hole is punched in the middle of the plate, and reinforced with masking tape. The cup-hangers are 2.5 ft. lengths of heavy wire, with a large hook bent at the top (for hanging over branches) and a small hook bent at the bottom (for attaching the bait cup and rain cap). To assemble the trap, one heavy rubber band is passed around the cup, near the top; another rubber band is looped through the first, and the free end then passed through a 1.5 inch length of rubber tubing, then through the hole in the paper plate, and finally hooked over the lower end of the wire hanger. This holds the paper plate over the cup in such a way that it keeps out the rain, and a good deal of the light, but does not cover the mouth. Flies are removed from the cup by the use of a pint Mason jar. Since some spray paints are insect repellent when fresh, the painting should be done well in advance of the anticipated trapping period.

Traut, H., and T. Mohr A simple and practical construction for bottling Drosophila culture medium. For the bottling (not preparation) of the culture medium we use an apparatus constructed according to the following principle which prevents the food from premature solidification during the filling procedure: the previously prepared medium is filled into a metal funnel, which is surrounded by a (commercial) "ring-heater for a water boiler". Regulation of the temperature is effected by a rheostst, heat isolation by asbestos. A stopcock allows the bottling of the medium in the desired portions. The apparatus is mounted in such a way that vials of different sizes can be filled. For better cleaning it is easy to dismantle.

Vloeberghs, J. V. A rapid method for preparing equal surface and volume food samples. For the exact comparison of biomass production of different D. melanogaster genotypes food samples of exactly equal volume and free surface are required. With the following method 80 to 90 food cups/hour fulfilling these requirements between error limits of 0.005 gr per cup can be prepared.

Normal hot culture medium is poured into a decantation cylinder with a ground stop-cock and neck (15 mm inner diameter). Through a perforated cork stoppering at the top, the cylinder is connected with a small air pump (aquarium type). A copper spiral with hot water flow can be twisted around the cylinder to keep its content warm and to avoid clogging of the neck. The apparatus is fixed above the plate of a very sensitive electronic balance, where the food cups are placed and weighed. The distance between cup and the outflow of the cylinder should be about 1 cm. Opening of the ground stop-cock allows the food to flow under pressure into the cup. The ground stop-cock is closed as the desired amount of food is attained. Free surface equality is guaranteed by using cups of equal diameter. In this laboratory flat bottomed, cylindrical glass cups of ± 25 mm depth and 26 mm inner diameter and attachable by means of Scotch tape to glass tubes of equal diameter are currently used.

PERSONAL AND LABORATORY NEWS

Alice Bull of Wellesley College is on leave for the year 1960-61. She is continuing her work on developmental genetics of Drosophila in Dr. Donald F. Poulson's laboratory at Yale University.

F. DeMarinis writes from Italy: "I am now on leave of absence from Fenn College and presently located at the American Embassy in Rome. Scientists desiring information on scientific meetings, aids, grants, fellowships and other problems in general that may involve the exchange of scientific knowledge between scientists in the two countries, can write to me: Office of the Scientific Attaché, American Embassy, Rome, Italy."

M. Dharmarajan, Department of Animal Genetics, Veterinary College, Madras 7, India, is engaged in a systematic and ecological survey of Drosophila in Madras. He would appreciate receiving reprints of papers on Drosophila, back numbers of DIS, and type specimens.

Allen S. Fox is spending 1961 as a Fulbright Research Professor in Australia and Israel. During the first six months of the year he will work at the Animal Genetics Division, CSIRO, Zoology Laboratory, Sydney University, Sydney, Australia. During the last six months he will be at the Weizmann Institute of Science, Rehovoth, Israel. In August he will attend the Fifth International Congress of Biochemistry at Moscow, USSR.

Kathryn E. Fuscaldo is now Assistant Professor, Department of Biology, Graduate School, St. John's University, Jamaica, New York.

Y. Hiraizumi will move from the Department of Genetics at Wisconsin University to the National Institute of Genetics, Misima, Japan, after December, 1960.

H. F. Hoenigsberg, formerly research fellow of the Italian committee for nuclear research, (CNRN, now CNEN) biology division, has been appointed professor of Genetics at the University of the Andes, Bogotà, Colombia. He would appreciate receiving reprints of papers on Drosophila, back numbers of DIS, and type specimens.

M. J. Hollingsworth is returning to the United Kingdom at the end of 1960 after five years at the Zoology Department, University College of Ghana. He has been appointed to a Lectureship at St. Bartholomew's Hospital Medical College, London, E. C. 1.

Aziz F. Khishin, formerly at the department of genetics of the University of Cairo, has been appointed Associate Professor at the new department of genetics of the University of Assuit, Egypt.

Rolf and Sabina Loosli, who have been at the Jackson Laboratory, have returned to the University of Zürich, Switzerland.

Charles G. Mead will join the Biology Division, Oak Ridge National Laboratory during February, 1961. He will continue his work on Drosophila DNA, begun at Michigan State University.

Dwight Miller has returned to his teaching and research activities at the University of Nebraska after a year at NSF.

Sidney Mittler, formerly with the Armour Research Foundation, has joined the Department of Biological Sciences, Northern Illinois University, DeKalb.

T. Mukai has moved from the Department of Biological Sciences at Purdue University in Indiana to the National Institute of Genetics, Misima, Japan.

Donald J. Nash has been appointed Assistant Professor of Genetics in the Department of Botany and Plant Pathology at Pennsylvania State University where he will direct the Drosophila laboratory. He would appreciate receiving available reprints, either old or new.

Y. K. Paik has moved from the Biology Department of National Chunnam University, Kwangju to the Department of Biology, College of Science and Engineering, Yonsei University, Seoul, Korea.

Dean Parker is on leave from the University of California at Riverside as Program Director for Genetic Biology at the National Science Foundation this year.

Sarah Bedichek Pipkin Please continue to mail reprints to 801 East 23 St., Austin Texas.

O. S. Reddi, who has spent 10 months working at the Institute of Animal Genetics under the direction of Dr. Auerbach on a post-doctoral Fellowship from the Nuffield Foundation, on chemical mutagenesis, will be returning to resume work at the Veterinary College, Osmania University, Hyderabad 7, India. He would appreciate receiving reprints and back numbers of DIS.

Gerda Rolfes, Institut für Tierzucht und Haustiergenetik, Giessen, Germany, was at the Animal Husbandry Department, Iowa State University, Ames, for the school year 1959-60 on a post-doctoral fellowship from the Kellogg Foundation. In addition to livestock breeding research she carried out a study on viability of superfemale Drosophila. A manuscript reporting her observations has been submitted to Jour. Heredity.

Seoul, Korea: The Department of Biology of Yonsei University is starting a new Genetics Laboratory of Drosophila. All reprints available, either old or new, will be gratefully received by Yong Kyun Paik.

Seoul, Korea: The Department of Biology of Sungkyun-Kwan University is starting a new Genetics Laboratory of Drosophila, where Y. K. Paik has been appointed as a consultant and a special lecturer.

B. L. Sheldon, of the CSIRO Animal Genetics Division, Animal Genetics Laboratory, Dept. of Zoology, Sydney University, is spending this year as a post-doctoral student in the Division of Biology, California Institute of Technology, Pasadena, California.

Azim O. Tantawy, of Alexandria University, Egypt, U.A.R., had returned to his country after spending two sabbatical years in the United States of America. He spent the first year working with Professor Dobzhansky, Th. of Columbia University and the other year at Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

MATERIALS REQUESTED OR AVAILABLE

Jack Bennett: "A SIMPLIFIED METHOD OF DROSOPHILA CULTURE FOR THE CLASSROOM" by Jack Bennett is available from: Educational Bulletin Service, Northern Illinois University, DeKalb, Illinois, for 10¢. It was written to answer requests from high school teachers and students on culture methods. Drosophila laboratories bothered by such requests are invited to refer their correspondents to the above address.

A. Chovnick and A. Schalet are currently engaged in a pseudoallelic investigation of the rosy locus (see note this issue). Over three dozen new rosy mutants, most of which are viable and fertile in homozygous condition, have been obtained by X-raying a cu kar chromosome. Because of the relative closeness of the kar ry linkage, almost all of the stocks are still homozygous for kar. Consequently, we are not prepared to describe the new rosy mutants at this time. We will be happy to provide information concerning these stocks and we would appreciate any information concerning any new rosy mutants obtained elsewhere.

Sara Frye (Dept. of Zoology, Indiana University, Bloomington, Indiana) would appreciate receiving yellow mutants recovered in the female and since 1960 of any size sample (the larger, the better) in any of the following categories:

1. of spontaneous origin in scute-8 or scute-S1 chromosomes
2. X-ray induced in normal chromosomes
3. X-ray induced in scute-8 or scute-S1 chromosomes of oogonia
4. U.V. induced in scute-8 or scute-S1 chromosomes
5. Neutron-induced in scute-8 or scute-S1 chromosomes

6. Chemically-induced in scute-8 or scute-S1 chromosomes
7. any dark yellow (y^2 phenotype) in scute-8 or scute-S1 chromosomes
8. any yellow allele that complements with y^2
9. any reverse mutants at the yellow locus in any X-chromosome, spontaneous or induced.

H. F. Hoenigsberg (Department of Genetics, University of the Andes, Bogotá D. E., Colombia, South America) would appreciate receiving reprints of papers on genetics. He would appreciate receiving the Payne Inversions, In(3L)P discovered in wild type D. melanogaster.

Edward C. Keller: The twenty-four sib-mated long inbred lines of D. melanogaster, derived from a wild population near State College, Pennsylvania, that are described in the stock list of University Park, Pennsylvania, are going to be discarded during the latter part of 1961. These lines have been reared at three different constant temperatures and there are 12 lines remaining at 26°C, 3 at 22°C, and 9 at 18°C. Temperature was the only selective mechanism involved in the derivation of these lines. If you have use for these lines or expect to use them contact Edward C. Keller, Jr., Buckhout Laboratory, University Park, Pennsylvania.

Aziz F. Khishin, The University of Assuit, Egypt, has started a new department of genetics. All available reprints will be much appreciated.

F. Mainx (Institut f. Allgemeine Biologie, University of Vienna, Wien IX. Schwarzspanierstr. 17) would appreciate obtaining strains of Megaselia scalaris (=Aphiochaeta xanthina) from different countries as well as strains of other species of Phoridae easily bred in the laboratory.

E. Novitski: A number of apparently precise reinversions of the rst^3 inversion, found in the F_1 of irradiated heterozygous females, may be had by any skeptical worker who is interested in examining these cases more closely. These five or six independent cases will be discarded in the near future.

Pauline B. Shapard: A series of D. melanogaster stocks which includes most of the combinations that can be made between each of a series of v alleles (v^1, v^2, v^{36f}, v^{48a}, v^{51c}, v^k) and the suppressor of vermilion (su^2-s) and the non-allelic suppressible mutants speck, purple, and sable, are available to anyone interested. They are currently being maintained by E. Novitski, University of Oregon, who plans to discard these stocks a few months after this DIS appears.

A. F. Yanders (Department of Zoology, Michigan State University) would be grateful for stocks of Drosophila species, especially exotic species, in which the ventral seminal receptacle and/or testes are unusually short (less than 1 mm) or unusually long (greater than 3 mm).

ANNOUNCEMENTS

Oster, Irwin I. Establishment of a second Drosophila Stock Center.

A National Science Foundation Grant for the establishment of a second Drosophila Stock Center has recently been awarded to The Division of Chemotherapy of The Institute for Cancer Research, Philadelphia 11, Pennsylvania. This additional Center, where emphasis will be placed on the maintenance of basic stocks in a manner similar to that employed by Professor E. B. Lewis at the Center in the California Institute of Technology, will 1) serve as a source of supply for virtually any research needs which might arise regardless of field of genetic interest, 2) provide insurance against loss of all or part of such a collection by some unforeseen catastrophe, 3) allow each center to make replacements for stocks which for one reason or another have broken down and 4) provide building blocks for the synthesis of the so-called "marker and tester stocks" and other useful combinations of mutations which can be used for research and teaching in genetics.

Some 2500 mutant strains will be maintained at the Center. These will include duplicates of the 800 varieties currently maintained by Professor E. B. Lewis at the Stock Center in Pasadena, 600 stocks representing the major portion of the strains maintained by Professor H. J. Muller at Indiana University, 200 strains presently maintained by Professor J. Schultz in the Department of Genetics of The Institute for Cancer Research, and 800 strains consisting of other useful mutations, combinations of them, and multiple alleles of loci of unusual interest obtained from other laboratories or synthesized by my group.

I would suggest that other workers should contribute potentially useful stocks for inclusion in the collection. The main requisites for maintenance of such strains will be that they are held in a combination not requiring continual selection and that they represent new loci, alleles of unusual interest, improved balancers, etc. In order to avoid the inclusion of too many stocks of questionable usefulness in the permanent collection, consultations with the other members of the Subcommittee on Drosophila Stocks composed of Professors E. Novitski and E. B. Lewis, among others, will be held from time to time concerning the advisability of adding any of these newly contributed stocks.

No stocks of the permanent collection will be discontinued (except when it has been lost by contamination and cannot be reconstructed from existing parts) without notice being given at least one year in advance and after consultation with the Drosophila Subcommittee.

The National Science Foundation Grant also provides for the publication and distribution, through Drosophila Information Service, of descriptions of the stocks and explanations for their use.

Contributions from H. J. Muller:

1. Two books in the Russian language published in Moscow within the past two years that are of potential interest to many of our readers are (to state their titles, etc., in English) "The Oxygen Effect in the Action of Ionizing Radiation," by E. S. Shchepotyeva, S. N. Ardashnikov, G. E. Lurie, and T. B. Rakhmanova, 1959, State Publishing House of Medical Literature, Moscow, 187 pp., and "Ionizing Radiation and Heredity," edited by N. P. Dubinin, 1960, Press of the Academy of Sciences of the U.S.S.R., Moscow, 344 pp. These works are on a high scientific level. They take into account and give abundant references not only to recent findings and discussions in Soviet countries but also to those reported in Western and Japanese-sponsored publications up to within three years of the issuance of the books.

The first mentioned book contains five chapters, dealing respectively with (1) the oxygen effect of radiation of low linear energy transfer, (2) that of radiation of high linear energy transfer, (3) peculiarities in the expression of the oxygen effect, (4) the utilization of the effect for deciding various important questions concerning the mechanism of the biological action of ionizing radiation, and (5) conclusions.

The second book, after a short forward by Dubinin, contains the following chapters:

A. A. Prokofyeva-Belgovskaya, "The structure of chromosomes." (53 pp.)

N. P. Dubinin, "The mechanism of the action of ionizing radiations on heredity." (43 pp.)

N. L. Deloné, "Chromosome rearrangements brought about by ionizing radiations." (19 pp.)

M. L. Belgovsky, "The dependence of the radiosensitivity of the chromosomes of animals on the stages of development of the germ cells." (32 pp.)

N. L. Deloné, "The sensitivity of different phases of mitosis and meiosis to ionizing radiations." (21 pp.)

B. N. Sidoroff and V. V. Khvostova, "Factors influencing the genetic effect of ionizing radiation." (32 pp.)

N. P. Dubinin and M. A. Arsenyeva, "Radiation genetics of man." (31 pp.)

S. Z. Mindlin, "Radiation selection of microorganisms." (33 pp.)

N. P. Dubinin, V. V. Khvostova and N. L. Deloné, "Ionizing radiation and selection in plants." (31 pp.)

D. D. Romashoff and K. A. Golovinskaya, "Radiation biology and genetics of fish." (19 pp.)

It is to be hoped that this book, with its diagrams and photographs of normal and abnormal chromosome structures and events, its diagram of the replication of the

Watson-Crick model, its numerous figures and tables and rich bibliographies, will become available in English before the rapid advances occurring in these fields cause it to become out of date. It is encouraging to learn of this activity in genetics on the part of our Russian colleagues.

2. Copied from Daedalus (Summer, 1960), pp. 597-598, article by Gustav Wetter:

". . . The June 1959 Plenary Session of the Central Committee of the Communist Party of the Soviet Union again gave extensive consideration to the problems of the organization of science. . . . Khrushchev objected to this (that Dubinin, the principal opponent of Lysenko, had been transferred from Moscow to the newly erected University of Novosibirsk in Siberia), accusing Dubinin of having contributed little to either theoretical or applied science, saying that he owed his renown entirely to his attacks on Lysenko, and that if his work in Moscow had been fruitless, it would be no less so in Novosibirsk or Vladivostok. Khrushchev did add that he did not wish to be in the position of umpire in the controversy between the two scientists, that the best judges are experience and life, and that these had decided in favor of the theory of Michurin and Lysenko."

3. Word has been received by H. J. Muller of the death on April 15, 1959, in Moscow, of Mark L. Belgovsky. He died unexpectedly, during his sleep, at the age of 53. He is survived by his wife, the cytogeneticist Alexandra Alexeyevna Prokofyeva-Belgovskaya, and by a grown son, a physicist.

Belgovsky is best known for his important works on the mechanism of production of translocations and of minute rearrangements, and on the basis of the mosaicism arising when a heterochromatic region is juxtaposed into the vicinity of a euchromatic one that was not previously in its neighborhood. Most of his work that has become known in the West was published in the decade 1936-1946. In 1957 he appeared as co-author, with M. A. Arsenyeva and others, of a work in Russian on radiation genetics in "Itogi Nauki, Biologicheskie Nauki, 1, Radiobiologia," p. 329-379, Press of Acad. of Sc. of U.S.S.R., Moscow and posthumously, in 1960, of a chapter in Russian, "The dependence of the radiosensitivity of the chromosomes of animals on the stage of development of the germ cells," in "Itogi Nauki, Biologicheskie Nauki, 3, Ionizing Radiation and Heredity," p. 123-154, Press of Acad. of Sc. of U.S.S.R., Moscow. During the period preceding his death he was actively at work on this subject and on the mechanism of mutation. Moreover, a letter written by him to Muller on Mar. 10, 1959, a few weeks before his death, tells of his continued interest in the problem of the nature of the gene and of his desire to keep up with the current literature on that subject.

Belgovsky was outstanding not only in his experimental and theoretical work in genetics but also in educating and inspiring both young investigators and contemporaries. He was warmly loved for his personal qualities, including his intellectual and moral integrity, the genuineness of his attachments, and his humanity.

Following a trip made to Moscow in 1960, Dr. Alexander Hollaender has brought back a list of geneticists working in the Laboratory of Radiation Genetics in Moscow headed by N. P. Dubinin. Besides Dubinin, head of the Laboratory, the list includes the following chief investigators:

Arsenyeva, M. A.
Glembozky, J. L.
Khvostova, V. V.
Prokofyeva-Belgovskaya, A. A.
Romaschoff, D. D.
Sakharoff, W.
Sidorov, B. N.
Sokoloff, N. N.
Sorokina, O. N.

There are in addition the following assistants:

Abeleve, E. A.
Beljaeva, V. V.
Bocharoff, U.
Mansurowa, W. W.
Shamina, Z.
Shawelson, R.
Sherbakoff, W.
Valena, S. W.

The address is Laboratory of Radiation Genetics
N. P. Dubinin, Lenin's Per.61/1, pl. 30,
Moscow, U.S.S.R.

Geographical

(Alphabetically arranged according to country, city, laboratory).

ARGENTINA

Buenos Aires
Comision Nacional de Energía Atomica, Departamento de Medicina y Biología, Seccion Genética

Ercolini, Luisa Susana. Research Assistant.
de Fincati, Wanda Pirovana, Ing. Agr. Research Student.
Kirschbaum, Werner F. Research Assistant.
Leon, Williams N. Technical Assistant.
Muñoz, Enzo Rubén. Research Student.
Paz, Bonifacia del Carmen. Technical Assistant.
Valencia, R. Marie. Ph.D. Chief of Genetics Section. Radiation genetics.

Buenos Aires
Universidad de Buenos Aires, Departamento de Biología, Laboratorio de Genetica y Citologia, Facultad de Ciencias Exactas y Naturales, Peru 222

Cacheiro, Nestor. Research Assistant.
Diez, Julio. Research Assistant.
Mazar-Barnet, B. Student Investigator. Chemical mutagenesis.
Valencia, J. I. Professor. Head of Department of Biology.

AUSTRALIA

Adelaide, South Australia
University of Adelaide, Department of Genetics.

Mayo, M. Jean. Ph.D. Lecturer
Hayman, D. L. Ph.D. Lecturer

Brisbane
The University of Queensland, Department of Zoology

Mather, Warton B. Ph.D. Senior Lecturer. Population Genetics.
Angus, D. B.Sc. Graduate student. Population Genetics.
Harlock, Rosalie. B.Sc. Graduate Student. Population Genetics.
Spurway, Rosalyne. Research Assistant.

Hobart, Tasmania
The University of Tasmania, Department of Zoology

Brink, N. G. B.Sc. (Hons.). Research student. Mutation.
Clark, A. M. Professor. Mutagenesis.
Clark, E. G. (Mrs.) Research Assistant.

Melbourne, Victoria
The University of Melbourne, Department of Zoology

Gunson, Mary M. M.Sc. Lecturer. Salivaries.
Strangio, V. A. M.Sc. Senior Demonstrator. Mutation.
Thomson, J. A. M.Sc. Lecturer. Population Genetics.
Wasserman, M. Ph.D. Senior Lecturer. Cytology; Evolution.

Sydney, New South Wales
Sydney University, CSIRO Animal Genetics Division, Animal Genetics Laboratory, Department of Zoology

Finlay, D. B.Sc.Agr. Experimental Officer.

Rendel, J. M. B.Sc., Ph.D. Chief of Division. Population Genetics. Selection.
Sheldon, B. L. B.Sc.Agr., Ph.D. Research Officer. Selection. Induction of mutations.

Sydney, New South Wales
University of Sydney, Department of Animal Husbandry

Barker, J. S. F. B.Agr.Sc. Lecturer. Population genetics.
Davey, Gillian B.Sc. Research Assistant. Population genetics.

AUSTRIA

Vienna IX (Wien IX)
Institut f. Allgemeine Biologie, Schwarzspanierstr. 17

Karlik, Anni Ph.D. Melanogaster: population genetics.
Kunze-Mühl, Elfriede Ph.D. Subobscura: cytogenetics.
Mainx, Felix M.D.,Ph.D. Professor. Head of department.
Neubert, Joseph Ph.D. Melanogaster: population genetics.
Ruderer, Elfriede Ph.D. Megaselia: genetics.
Ruttner, Friedrich M.D.,Ph.D. Genetics of the honey bee.
Sperlich, Diether Ph.D. Subobscura: population genetics.
Springer, Robert Ph.D. Megaselia: genetics.

BELGIUM

Louvain
The University, Agricultural Institute, Laboratory of General Genetics

Deconinck, C. V. Technical Assistant.
Elens, A. A. Ph.D. Guest Investigator. Sexual isolation.
Heuts, M. J. Professor. Genetics; population genetics.
Laureys, M. J. Technical Assistant.
Lints, F. A. Ph.D. Chargé de Recherches au F.N.R.S. Cytoplasmic inheritance.
Merckx, F. E. Technical Assistant.
Mouravieff, A. N. Technical Assistant.
Stiers, R. O. Curator of Stocks.
Vloeberghs, J. V. Technical Assistant.
Wattiaux, J. M. Graduate Student. Isolation mechanism.

BRAZIL

São Paulo
Universidade de São Paulo, Faculdade de Filosofia, Ciências e Letras - Departamento de Biologia Geral - Caixa Postal 8.105

Breuer, M. E. Technical Assistant. Comparative studies on Drosophila genitalia. Radiation genetics.
Cestari, A. N. Lic. Cien. Graduate student. Tissue culture. Human chromosomes.
da Cunha, A. B. Ph.D. Assistant Professor. Population genetics: polymorphism in Drosophila and effects of radiation on populations.
Frota-Pessoa, O. Ph.D. Assistant Professor. Heterosis in Drosophila. Human genetics.
Grzegorzewska, M. D. Graduate student. Speciation in Drosophila.
Lindsley, D. L. Visiting Professor. National Science Foundation Senior Postdoctoral Fellow.
Magalhães, L. E. de Ph.D. Assistant Professor. Population genetics and Speciation.
Pavan, C. Ph.D. Professor. Head of Department. Population genetics, radiation genetics and cytogenetics.
Pinto, S. C. Technical Assistant. Tissue culture: human chromosomes.
Souza, H. M. L. Lic. Cien. Assistant Population genetics, radiation genetics.
Takatsu, Y. Graduate student. Population genetics, radiation genetics.
Toledo, J. S. Lic. Cien. Assistant. Population genetics, radiation genetics.

Toledo, S. A. Lic. Cien. Assistant. Population genetics, radiation genetics.
Wajntal, A. Graduate student. Human genetics.
Wallace, B. Visiting Professor. National Institute of Health, U.S. Public Health Service, Research Fellow. Radiation genetics.

CANADA

Toronto
University of Toronto, Department of Zoology

Butler, L. Director of the Laboratory. Population genetics.
Seiger, M. B. Quantitative inheritance.

CHILE

Santiago
Universidad de Chile, Instituto de Biología "Juan Noé", Cátedra de Biología, Zañartu 1042

Brncic, D. Associate Professor. Population genetics.
Casanova, Adriana Technical Assistant. Curator of Stocks.
Covarrubias, Edmundo M.D. Research Assistant.
del Solar, Eduardo Graduate student. Population genetics.
Fernandez, Raúl Graduate student. Isolating mechanisms.
Koref-Santibañez, Susi M.D. Research Associate. Population genetics, isolating mechanisms.
Pellicer, M. Dolores Technical Assistant.

COLOMBIA

Bogotá
University of the Andes, Department of Biology, Apartado Aereo 4976

Hunter, Alice S. Ph.D. physiology, taxonomy - Drosophila.
Newball Assistant.

DENMARK

Copenhagen
University of Copenhagen, Institute of Genetics, 2A Øster Farimagsgade

Andersen, B. (Mrs.) Technical Assistant.
Frydenberg, Ove Lecturer, Population genetics, taxonomy.
Henningsen, K. Graduate student.
Sick, Knud Research Assistant. Population genetics.
Westergaard, M. Professor. Head of Department.

FINLAND

Turku
University of Turku, Institute of Genetics

Hannah-Alava, Aloha (Mrs.) Ph.D. Research Associate. Melanogaster: developmental genetics, mutations.
Heinonen, Pirkko (Miss) Research Assistant. Melanogaster: mutations.
Juvonen, Vuokko (Miss) Ph.Kand. Research Assistant, Melanogaster: interchromosomal effects.
Lehtonen, Aila (Miss) Research Assistant. Melanogaster: mutations.
Oksala, T. A. Ph.D. Professor. Head of Department. Melanogaster: activity of heterochromatin, interchromosomal effects.
Petâjâ, Terttu (Mrs.) Ph.Kand. Research Assistant. Melanogaster: interchromosomal effects.
Puro, J. Ph.Kand. Assistant Teacher. Melanogaster: mutations.

Savolainen, Salme (Mrs.) Technical Assistant.

FRANCE

Gif-sur-Yvette (S. et O.)
Centre National de la Recherche Scientifique, Laboratoire de Génétique évolutive et de Biométrie

Bergerard, J. Professor. Cytogenetics.
Besson (Miss) Graduate student. Population genetics.
Bigler, J. (Miss) Technician.
Bösiger, E. Ph.D. Chargé de recherches. Population genetics.
Duchemin, M. (Miss) Graduate student. Cytogenetics.
Laugé, G. (Miss) Assistant. Cytogenetics.
Leboulleux, P. (Mrs.) Technician. Curator of stocks.
Louis, M. (Mrs.) Technician.
Morio, H. Graduate student. Cytogenetics.
Piva, A. Graduate student. Quantitative inheritance.
Queiroz, J. (Mrs.) Stagiaire de recherches. Quantitative inheritance.
Tassencourt (Mrs.) Statistician.
Teissier, G. Professor. Head of Department. Population genetics; quantitative inheritance; biometry.

Gif-sur-Yvette (S. et O.)
Centre National de la Recherche Scientifique, Laboratoire de Génétique Formelle

Brun, G. Chef de travaux pratiques. CO_2 sensitivity in Drosophila.
Lestrange, M.-Th. de (Miss) Attachée de recherches. CO_2 sensitivity in Drosophila.
L'Héritier, Ph. Professor. Head of Department. CO_2 sensitivity in Drosophila.
Ohanessian-Guillemain, A. (Mrs.) Attachée de recherches. " " "
Plus, N. (Mrs.) Chargée de recherches. CO_2 sensitivity in Drosophila.
Proust, J. (Mrs.) Attachée de recherches. Quantitative inheritance in Drosophila.
Vigier, Ph. Maître-assistant. CO_2 sensitivity in Drosophila.

Lyon. Laboratoire de Zoologie Experimentale. See Page 155.

Paris
Faculté des Sciences, Laboratoire de Zoologie, 1 rue Victor Cousin, Paris 5ème

de Frescheville, J. Chef de travaux. Cytogenetics.
Guillaumin, M. Assistant. Quantitative inheritance.
Melou, J. P. Assistant. Quantitative inheritance.
Orillard, C. Graduate student.
Petit, C.(Mme) Chef de travaux. Quantitative inheritance and sexual isolation.
Teissier, G. Professor. Head of Department. Population genetics; quantitative inheritance; biometry.
Usciati, J. (Miss) Assistant. Quantitative inheritance.

Strasbourg (Bas-Rhin)
Université de Strasbourg, Faculté des Sciences

Digot, A. Maître de Conférences. CO_2 sensitivity in Drosophila.

GERMANY

Berlin-Dahlem
Institut für Genetik der Freien Universität Berlin, Rudeloffweg 9

Belitz, Hans-Joachim (Dr.) Research Assistant. Melanogaster: induced mutations.
Bochnig, Veronika (Dr.) Research Assistant. Melanogaster: physiological genetics, radiation genetics.
Halfer, Carla (Dr.) Research guest. Melanogaster: radiation genetics.
Kotte, Jutta Technical Assistant. Melanogaster: radiation genetics.
Kromm, Natalie Technical Assistant. Curator of stocks, chemogenetics.
Lüers, Herbert (Prof. Dr.) Director. Comparative genetices; mutagens.

Lüers, Thea (Mrs., Dr.) Guest Associate. Drosophila neurology.
Nöthel, Horst Graduate student. Radiation genetics.
Pasternak, Luise Graduate student. Melanogaster: DDT-resistance.
Polzin, Walter Technical Assistant. Radiation genetics.
Röhrborn, Gunter (Dr.) Research Assistant. Drosophila tumors; chemical mutagens.
Rudolph, Edeltraud Technical Assistant. Melanogaster: cytology.
Struck, Eva (Mrs., Dr.) Research Assistant. Insects: cytology.
Winterfeld, Giesela Graduate student. Melanogaster: radiation genetics.
Wolf, Erich (Dr.) Associate. Insects: cytology.

Berlin-Buch
Institut für Medizin und Biologie, Genetische Abteilung

Leppin, Helga Technical Assistant. Curator of stocks.

Hamburg-Eppendorf
Universitäts-Frauenklinik, Strahlenbiologische Abteilung

Henke, Heinz Dr. Assistant.
Höhne, Günter Dr.habil. Assistant.
Künkel, Hans A. Dr. Assistant.
Schubert, Gerhard Prof.Dr. Director. Melanogaster: radiation genetics; chemical mutagens.
v.Volkmann, Irene Technical Assistant.

Hamburg 13
Zoologisches Staatsinstitut und Zoologisches Museum, Bornplatz 5

Koske-Westphal, Th. Ph.D. Study of hybrids between triploid melanogaster females and X-rayed simulans males.
Kosswig, Curt Prof.Dr. Director.

Heidelberg
Institut fur Strahlenbiologie, Kernforschungszentrum Karlsruhe

Apitzsch, Ursula Curator of Stocks.
Catsch, Alexander Prof.Dr. Drosophila genetics.
Ebeling, Wolfgang Graduate student. Drosophila genetics.
Hotz, Gerhart Dr. Bacteriophage genetics.
Köhnlein, Wolfgang Dipl.Phys. Radiation biology .
Miltenburger, Herbert Dr. Neurospora genetics.
Müller, Adolf Dr. Radiation biology.
Traut, Horst Dr. Drosophila genetics.
Zimmer, Karl Günther Prof.Dr. Radiation genetics

Heidelberg
Universität Heidelberg, Zoologisches Institut, Sofienstr. 6

Duspiva, Franz Dr. Director. Biochemistry.
Hagens, Hans-Wolfgang Dr. Assistant. Physiology.
Ludwig, Herbert Dr. Assistant. Insect metabolism.
Wette, Reimut Dr. Biometry, genetics.
Zebe, Ernst Dr. Assistant. Muscular physiology.

Marburg a.d.Lahn
Zoologisches Institut der Universität

Becker, Gweneth L. Ph.D. Independent investigator.
Becker, Hans J. Ph.D. Assistant.
Buchholtz, Christiane Ph.D. Assistant.
Scriba, Martin Graduate student.
Seidel, Friedrich Ph.D. Professor. Head of Department.

Mariensee über Neustadt a.Rbge.
Max-Planck-Institut für Tierzucht und Tierernahrung

Gottschewski, G. H. M. Prof.Dr. Head of Department. Developmental and physiological genetics.
Querner, Waltraud Dr. Assistant Stockkeeper. Tissue culture.
Schwinck, Ilse Dr. Assistant. Physiological genetics.
Zimmermann, Wolfgang Dr. Assistant. Genetics.

Tübingen
Max-Planck-Institut für Biologie

Beermann, W. Dr. Director.
Hess, O. Dr. Research Assistant.
Seidel, S. (Miss) Research Assistant. Graduate student.

GHANA

Legon, Accra.
University College of Ghana, Department of Zoology

Hollingsworth, M. J. Lecturer. (see personal news)
Salitey, A. S. Curator.

Legon, Accra.
University College of Ghana, Department of Chemistry

Blair, J. A. Lecturer. Origin of pteridine compounds in Drosophila.

GREAT BRITAIN

Aberdeen, Scotland
University of Aberdeen, Department of Zoology

Begg, C. M. M. Dr. Lecturer. Drosophila: developmental genetics; tissue culture.
Cruickshank, W. J. Graduate student. Drosophila: tissue culture.
Pringle, C. R. Dr. Assistant. Drosophila: tissue culture. Paramecium: antigens.

Birmingham, 15, England
The University, Department of Genetics

Cooke, Patricia Assistant Experimental Officer. Selection and competition.
Lawrence, M. J. Ph.D. Lecturer. Recombination and selection.
Mather, K. D.Sc. F.R.S. Professor. Selection and competition.

Cambridge, England
Cambridge University, Department of Genetics

Alderson, T. Ph.D. Research Worker. Chemical mutagenesis.
Gibson, J. B. Assistant in Research. Selection.
Graham Brown, W. D. Research student.
Nash, D. Research student.
Parsons, P. A. Ph.D. Demonstrator. Population genetics, competition.
Pelekanos, M. Research student.
Spickett, S. G. Research student.
Thoday, J. M. Ph.D. Professor. Selection, particularly disruptive.
Wolstenholme, D. R. Research student. Disruptive selection.

Chalfont St. Giles, Bucks, England
Institute of Cancer Research: Royal Cancer Hospital, Pollards Wood

Benyon, E. Technical Assistant.
Fahmy, Myrtle J. Ph.D. Mutagenesis.

Fahmy, O. G. M.Sc.,Ph.D. Cytogenetics.
Hope, L. Technical Assistant.
Miller, M. Research Assistant.
Sweron, M. Technical Assistant.
Whurr, A. Technical Assistant.

Edinburgh 9, Scotland
Agricultural Research Council Poultry Research Centre, King's Buildings

Burnet, B. B.Sc. Research Assistant.
Sang, J. H. Ph.D.,F.R.S.E. Drosophila nutrition and physiological genetics.

Edinburgh, Scotland
Institute of Animal Genetics

Alexander, M. Radiation population genetics. N.I.H.-N.C.I. Fellow.
Auerbach, C. D.Sc.,F.R.S. Reader. Chemical mutagenesis.
Basden, E. B. Research Assistant. Wild species.
Clayton, G. Lecturer. Selection.
da Silva, J. M. Selection limits.
Kelsall, P. J. Graduate student. Non-disjunction.
Knight, G. R. Research Assistant. Subobscura salivaries.
Misra, R. K. Ph.D. Allometry.
Perry, M. Research Assistant. Autoradiography.
Reeve, E. C. R. D.Phil. Quantitative inheritance.
Robertson, A. D.Sc. Quantitative genetics.
Robertson, F. W. D.Sc. Population and physiological genetics.
Royes, V. Graduate student. Drosophila nutrition.
Sen, B. K. Graduate student. Quantitative genetics.
Slizynska, H. Ph.D. Salivaries.
Slizynski, B. M. Ph.D. Salivaries.
Waddington, C. H. Sc.D.,F.R.S. Professor. General genetics.

Glasgow, W.2., Scotland
The University, Department of Genetics

Forbes, E. C. Chief Technician
Pontecorvo, G. F.R.S. Professor.

Harwell, England
Medical Research Council, Radiobiological Research Unit, Harwell, Didcot, Berks

Allen, C. Technical Assistant.
Jempson, J. Technical Assistant.
Lamerton, M. Technical Assistant.
McSheehy, T. W. B.Sc. Research Assistant.
Purdom, C. E. Ph.D. Radiation Genetics.

London, W.C.1, England
University College, Department of Biometry, Eugenics and Genetics

Gruneberg, H. Professor.

London, W.C.1, England
University College, Department of Zoology

Clarke, Jean M. Research Assistant.
Maynard Smith, J. Lecturer.
Sondhi, K. C. Research Assistant.

Manchester, England
Christie Hospital and Holt Radium Institute, Cytogenetics Laboratory

Bateman, Angus J. Radiation mutagenesis.
Chandley, Ann C. Radiation mutagenesis.

Manchester 13, England
The University, Departments of Botany & Zoology

Hartshorne, J. N. Lecturer in genetics.

Nottingham, England
The University, School of Agriculture, Department of Agricultural Sciences, Sutton Bonington, Loughborough, Leics.

Kheiralla, A. Research student.
Peat, W. E. Research student.
Whittington, W. J. Ph.D. Lecturer.

Sheffield, 10, England
The University, Department of Genetics

Boam, T. B. Chief Technician.
Beardmore, J. A. Lecturer. Homeostasis, disruptive selection.
Clutterbuck, A. J. Research student.
Roper, J. A. Professor. Microbial genetics.

INDIA

Calcutta - 19
Calcutta University, Department of Zoology, Cytogenetics Laboratory, 35, Ballygunge Circular Road.

Banerjee, S. K. B.Sc. Graduate student. Ananassae: chromosome map, male crossing-over.
Ghosh, H. B.Sc. Laboratory Technician & Curator of Stocks.
Mitra, J. B.Sc. Graduate student. Ananassae: male crossing-over, chromosome map.
Ray-Chaudhuri, S. P. Ph.D.,F.N.I. Reader. (On leave). Melanogaster & ananassae; radiation genetics, chemical mutagenesis.
Sarkar, S. K. M.Sc. Research worker. Ananassae; melanogaster.

Calcutta - 35
Indian Statistical Institute, 203, Barrackpore Trunk Road

Haldane, J. B. S. Research Professor.
Jayakar, S. D. Research Scholar.
Mukherjee, M. Technician.
Spurway, H. Reader.

Madras 7
Veterinary College, Department of Animal Genetics, Vepery

Dharmarajan, M. M.A., M.Sc.,Ph.D. Head of Department. Drosophila species.
Narayana Rao, N. M.A. Assistant. Melanogaster: species, ecology.
Suguna, S. G. M.Sc. Advanced student. Drosophila mutagenesis.

ISRAEL

Jerusalem
Hebrew University of Jerusalem, Department of Zoology

Barak, Elisheva Research student. Induced inversions.
Bernstein, Naomi M.Sc. Research student. Induced inversions.
Bloch, Navah Research student. Pteridines: position effect. Human genetics.

Eckstein, Edna M.Sc. Assistant. Mosquito genetics.
Fattal, S. Laboratory Assistant.
Friedlander, Michael Research student. Cytology.
Goldschmidt, Elisabeth Ph.D. Associate Professor. Pteridines: inversion in natural populations. Human genetics.
Hurvitz, Dalia Research student. Pteridines.
Lederman-Klein, Ada M.Sc. Assistant. Homeotic mutant.
Levine, Ora Research student. Fluorescent substances in hybrids.
Rapaport, Sarah Laboratory Assistant.
Ritte, U. Research student. Rodents: population dynamics.
Ronen, A. M.Sc. Assistant. Crossing over. Human genetics.
Shahaph, Tamar Research student. Cytology.
Wahrman, J. Ph.D. Lecturer. Population dynamics.
Yarkoni-Freund, Ora M.Sc. Assistant. Cytology.

ITALY

Milano
Università di Milano - Istituto di Genetica, Via Celoria 10

Barigozzi, C. D.Sc. Professor of genetics. Director. Genetics of melanotic tumors.
Castiglioni, M. C. D.Sc. Assistant. Developmental genetics. Tissue culture.
di Pasquale, A. D.Sc. Assistant. Cytoplasmic inheritance.
Gallucci, E. M.D. Research Fellow. Induced mutations.
Giavelli, S. M.D. Research Fellow. Induced mutations.
Halfer, C. D.Sc. Assistant. Genetics of melanotic tumors.
Hoenigsberg, H. F. B.Sc.,D.Sc. Research Fellow. Courtship in Drosophila. Biochemical genetics. Induced mutations.
Locatelli, F. Technician. Curator of Stocks.
Pozzi, L. D.Sc. Research Fellow. Induced mutations.
Rezzonico, Raimondi G. D.Sc. Assistant. Experimental cytology.
Sgorbati, G. D.Sc. Assistant. Genetics of melanotic tumors.
Sironi, G. P. Student Assistant. Induced mutations.

Napoli
Dell Università, Istituto di Genetica

Carfagna, M. Research Associate.
Cioffi, E. Research student.
De Capoa, A. Research Associate.
Megna, F. Curator of Stocks.
Montalenti, G. Director (will move to Rome, Istituto di Genetica, Facoltà di Scienze in the course of 1961).
Nicoletti, B. Research Associate (attached to the Istituto di Genetica, Facoltà di Scienze, Università di Roma).
Olivieri, G. Research student.
Solima, A. Research Associate. (Pro-tempore in the Department of Zoology, Columbia University, New York, N.Y., U.S.A.).
Virzo, A. Research student.

Pavia
Università di Pavia, Istituto di Genetica, via Sant'Epifanio 14

Buzzati-Traverso, A. A. Professor. Director.
Canuti, N. Technical Assistant.
Gerletti, M. Graduate student.
Ghini, C. Research Assistant.
Guglielmi, A. Technical Assistant.
Magni, G. E. Professor.
Peschiera, G. Curator of stocks.
Rasmussen, I. E. Assistant.
Ritossa, F. Graduate student.
Scossiroli, R. E. Professor.

JAPAN

Anzyo-Shi, Aichi
Nagoya University, Faculty of Agriculture, Laboratory of Animal Breeding

Bito, J. Graduate student. Melanogaster; mutation.
Esaki, K. Assistant. Melanogaster; mutation.
Kondo, K. Dr., Professor. General genetic problems.
Nozawa, K. Instructor. Melanogaster, other species; population genetics, mutation, statistics.
Ota, N. (Miss) Technical Assistant. Curator of stocks.
Tomita, T. Instructor. Melanogaster; mutation.

Chiba-shi
National Institute of Radiological Sciences, Biology Division

Inagaki, Eiichi Drosophila and Bombyx; mutation problems.
Kasai, Ichiko Technical Assistant.
Nakao, Yoshio Head of Division. Drosophila and Bombyx; physical and chemical mutagenesis.
Shiomi, Mioko Stock keeper.
Shiomi, Toshio Chief of Laboratory. Physiological genetics; mutation and lethal factors.
Tachibana, Hiroko Mutation problems.
Yasuda, Norikazu Population genetics; theoretical study.

Hiroshima
Hiroshima University, Faculty of Science, Zoological Laboratory

Minamori, S. Dr. Assistant Professor. Melanogaster: speciation.

Kobe
Kobe University, Biological Laboratory

Fujii, S. Dr.,Professor. Chromosomal aberrations; salivary chromosomes; developmental genetics.
Kanehisa, T. Research Assistant. Biochemical genetics of tumors.
Kawabe, M. Assistant Professor. Developmental genetics; variations.
Kitazume, Y. Research assistant. Cytochemical studies of lethal mutations.

Kyoto
Kyoto University, Faculty of Science, Department of Zoology

Imaizumi, Tadashi Assistant. Physiological genetics and embryology.
Kato, Masaru Dr.,Assistant Professor. Biochemical genetics and embryology. (Present address: Department of Poultry Sciences, Washington State College, Pullman, Washington, U.S.A.)
Kato, Mikio Dr. Research Associate. Biochemical genetics.
Miyoshi, Yasuhiro Graduate student. Physiological genetics.
Motobu, Hirotetsu Research Associate. Physiological genetics.
Nakamura, Kenji Dr.,Professor. Cytogenetics and physiology.
Okuda, Chizuko (Miss) Technical Assistant. Curator of stocks.

Misima, Sizuoka-ken
National Institute of Genetics

Hiraizumi, Y. Dr. Research Member (from December, 1960) Population genetics.
Imai, Y. (Miss) Technical Assistant.
Iyama, S. Dr. Research Member. Population genetics; competition and migration.
Kimura, M. Ph.D. Research Member. Population genetics; theoretical.
Masuda, H. (Miss) Technical Assistant.
Mukai, T. Ph.D. Research Member. Population genetics; radiation and polygene.
Nakamura, K. (Miss) Technical Assistant.
Narise, T. Research Member. Population genetics; competition and migration.
Nawa, S. Dr. Research Member. Biochemical genetics; pteridine and nucleic acid.
Nonami, K. (Miss) Technical Assistant.

Oshima, C. Dr. Head of Department. Population genetics; resistance, radiation and deleterious genes in natural populations.
Sakaguchi, B. Dr. Research Member. (Yale Univ.) Biochemical genetics; enzymes.
Sakai, K. Dr. Head of Department. Population genetics; competition and migration.
Taira, T. Research Member. Biochemical genetics; eye pigment formation and metamorphosis.
Tsukamoto, H. (Miss) Technical Assistant.
Yamada, Y. Dr. Research Member. Population genetics; mutation and selection.

Mitaka, Tokyo
International Christian University, Department of Biology

Kaminishi, H. Research Fellow.
Shoki, T. Instructor.
Sinoto, Y. Professor. Dean of College of Liberal Arts. Head of Department.

Okamoto, Kobe
Konan University, Biological Laboratory

Inouye, I. Research Associate. Melanogaster, selection.
Kaji, S. Dr.,Assistant Professor. Melanogaster: selection, physiological genetics.
Takaya, H. Dr.,Professor. Melanogaster: selection.

Osaka
Osaka University, Faculty of Medicine, Department of Genetics; and Osaka University, Faculty of Science, Biological Institute

Baba, Y. (Miss) Assistant. Musca: mutations and chromosomes.
Fujio, Y. Graduate student. Drosophila: embryological genetics.
Hiraga, S. Graduate student. Musca: mutations and resistance to insecticides.
Hiroyoshi, T. Graduate student. Musca: mutations and resistance to insecticides.
Ichioka, S. (Miss) Technical Assistant. Curator of Stocks.
Kikkawa, H. Dr.,Professor. Drosophila and Musca: chemical genetics and resistance to insecticides.
Kuroda, Y. Dr. Lecturer. Drosophila: embryological genetics.
Nobuki, R. (Miss) Technical Assistant. Curator of Stocks.
Ogita, Z. Assistant. Drosophila and Musca: chemical genetics and resistance to insecticides.
Seki, T. Lecturer. Drosophila: chemical genetics.
Tsukamoto, M. Assistant. Drosophila and Musca: chemical genetics and resistance to insecticides.
Watanabe, H. (Mrs.) Student. Drosophila: chemistry of resistance problems.

Sakai, Osaka
University of Osaka Prefecture, Department of Biology

Ogaki, M. Dr.,Assistant Professor. Melanogaster and Musca: physiological genetics.
Tanaka, E. Assistant. Melanogaster: physiological genetics. (On leave in Urbana, Illinois, U.S.A.)

Sapporo
Hokkaido University, Faculty of Science, Department of Zoology

Kaneko, A. Research Assistant. Geographical distribution; cytogenetics.
Makino, S. Dr.,Professor. Cytogenetics; population genetics.
Momma, E. Dr.,Assistant Professor. Geographical distribution; cytogenetics; population genetics.
Shima, T. Research Assistant. Geographical distribution.
Takada, H. Research Assistant. (Genetics Foundation of the University of Texas until August, 1961) Geographical survey; taxonomy.
Toyofuku, Y. (Mrs. Tonomura) Research Assistant. Geographical distribution; population genetics; cytogenetics.

Tokyo
Tokyo Metropolitan University, Faculty of Science, Department of Biology, Setagaya-ku

Akita, Y. K. Dr.,Professor. Melanogaster: gene action, biochemistry, radiation biology.
Fukatami, A. Graduate student. Melanogaster, other species: population genetics, sexual isolation.
Ikeda, H. Graduate student. Bifasciata, other species: population genetics, sex-ratio.
Kitagawa, O. Dr. Research Assistant. Bifasciata, melanogaster: population genetics, radiation genetics.
Kurokawa, H. Lecturer. Auraria, other species: population genetics, taxonomy.
Moriwaki, D. Dr.,Professor. Ananassae, bifasciata, melanogaster: population genetics, gene analysis, radiation genetics.
Ohba, S. Assistant Professor. Melanogaster, other species: population genetics, ecology, tumor.
Ohnishi, E. Dr. Research Assistant. Melanogaster, virilis: biochemistry.
Okada, T. Dr.,Professor. Various species: variations, taxonomy, ecology.
Tobari, I. Graduate student. Melanogaster: radiation genetics.
Tobari (Nakajima), Y. Research Assistant. Ananassae, other species: population genetics, heterosis, gene analysis.
Yoshida, Y. H. Research Assistant. Melanogaster, other species: population genetics, radiation genetics, statistics.

KOREA

Kongju, Chung Cheong Nam Do
Kongju National Teachers' College, Department of Biology

Chang, S. U. Research Assistant. Geographical survey.
Kim, O. S. Lecturer. Geographical survey.
Lee, T. J. Associate Professor. Population genetics, geographical distribution.

Kwangju, Chunnam
National Chunnam University, College of Liberal Arts & Sciences Department of Biology

Kim, D. U. Assistant Professor. Microbial genetics.
Kim, K. W. Assistant Professor. Dorosphila taxonomy.
Park, M. S. Instructor.
Wui, I. S. Instructor.

Seoul
Seoul National University, Department of Biology

Chung, Ok Ki Instructor. Cytogenetics.
Kang, Yung Sun Professor. Cytology.
Lee, Hei Yung Instructor. Cytogenetics.

Seoul
Sungkyun-Kwan University, College of Liberal Arts & Sciences, Department of Biology, Laboratory of Genetics

Kim, Min Cha (Miss) Undergraduate assistant.
Kim, Tok Seng Graduate Research Assistant.
Oh, Woo Cha (Miss) Undergraduate assistant.
Paik, Y. K. Dr. Consultant. (Permanent address: Yonsei University, Department of Biology, Seoul.
So, Woong Yung Graduate Research Assistant.
Sung, Ki Chang Graduate student.

Seoul
Yonsei University, College of Science & Engineering, Department of Biology

Kang, B. K. Graduate student. Radiation effects.

Paik, Y. K. Dr.,Associate Professor. Population genetics; radiation effects.

NETHERLANDS

Leiden
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Bonnier, G. Ph.D. Professor Emeritus. Melanogaster: viabilities of homozygotes and heterozygotes in populations. Persistency of autosomal lethals.
Caspersson, G. Research Assistant. Melanogaster: mutations.
Eiche, A. Ph.K. Research Assistant. Melanogaster: mutations, persistency of lethals.
Lindqvist, B. Research Assistant. Melanogaster: mutations.
Lüning, K. G. Ph.D. Director of the Institute. Melanogaster: mutations.
Montelius, I. Ph.K. Research Assistant. Melanogaster: population genetics.
Ramel, C. Ph.Lic. Research Assistant. Melanogaster: interchromosomal effects.
Savhagen, R. Ph.Lic. Research Assistant. Melanogaster: radiation effects on spermiogenesis.
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Johansson, K. Ph.M. Research Assistant. Melanogaster: selective mating.
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Rasmuson, Marianne Ph.D. Research Associate. Melanogaster: selection, population genetics.
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Chen, Pei Shen Ph.D.,Professor. Physiology and development.
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Diem,Claudia Graduate student. Peptides.
Faulhaber, Ilse Ph.D. Assistant. Lethals.
Gandolla, Maria Assistant. Nucleic acids of lethals.
Gloor, Regula Assistant. Lethals.
Goetz, Walter Graduate student. Salivaries of Drosophila subobscura.
Greuter, Mark Graduate student. Population genetics.
Hadorn, Ernst Ph.D.,Professor. Developmental and biochemical genetics; lethals.
Kaufman, Seymour Ph.D. Research guest. Biochemical genetics.
Luchsinger, Senta Graduate student. Malpighian tubes.
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Schläpfer, Theo Graduate student. Imaginal discs.
Schnitter, Marco Assistant. Lethals.
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Glass, Suzanne S. M.A. Research Assistant. Melanogaster: genetic control of tryptophan metabolism in Drosophila and its relation to abnormal growth.
Laufer, Hans. Ph.D. Assistant Professor. Differential gene action during development.
Mahowald, Anthony P. B.S. Graduate student. Developmental cytology of Drosophila.
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Ritterhoff, Rebecca K. B.S. Research Staff Assistant. Melanogaster: comparative study of induced mutation in males and females; effect of very low doses of ionizing radiation; Minutes; recessive lethals and spontaneous visibles in males and females; effects of oxygen concentration.
Warfield, Susan L. B.A. Research Assistant. Melanogaster: comparative study of induced mutation in males and females.
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House, Verl L. Ph.D. Associate Professor. Melanogaster: gene interaction; development of venation.
McClanahan, Charlene B.S. Graduate student. Melanogaster: temperature sensitive periods.
Plaine, Henry L. Ph.D. Associate Professor. Melanogaster: gene action of suppressor erupt and suppressor-tumor systems; high mutating system in a Swedish-b erupt strain.
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Capek, Ronald B.S. Selection, behavioral resistance to insecticides.
Dichter, Joan B.S. Research Technician.
Grimes, Jack B.S. Insecticide Resistance.
Landy, Ronald B.S. Wild populations.
Mittler, Sidney Ph.D. Tumors.
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Fujio, Y. Japan, Osaka
Fukatami, A. Japan, Tokyo
Fung, S.T.C. Ames, Iowa
Fuscaldo, K.E. Jamaica, N.Y.
Fusté, M. Spain, Barcelona
Futch, D.G. Austin, Texas

Gahne, B. Sweden, Uppsala
Gallucci, E. Italy, Milano
Gandolla, M. Switzerland, Zürich
García-Bellido, A. Spain, Madrid
Gardner, E.J. Logan, Utah
Gay, H. Cold Spring Harbor, N.Y.
Geer, B.W. Davis, Calif.
Gerletti, M. Italy, Pavia
Gersh, E.S. Chicago, Ill.
Gerstenberg, V.L. Austin, Texas
Ghini, C. Italy, Pavia
Ghosh, H. India, Calcutta
Giavelli, S. Italy, Milano
Gibson, J.B. Gr. Britain, Cambridge
Gidholm, K. Sweden, Uppsala
Gill, K.S. New Haven, Conn.
Gillies, G. Cold Spring Harbor, N.Y.
Glass, H.B. Baltimore, Md.
Glass, S.S. Baltimore, Md.
Glassman, E. Chapel Hill, N.Carolina
Gloor, H.J. Netherlands, Leiden
Gloor, R. Switzerland, Zürich
Goetz, W. Switzerland, Zürich
Goldschmidt, E. Israel, Jerusalem
Gottlieb, F. Berkeley, Calif.
Gottschewski, G.H.M. Germany, Mariensee
Gowen, J.W. Ames, Iowa
Graham Brown, W.D. Gr. Britain, Cambridge
Green, M.M. Davis, Calif.
Greenberg, R. Madison, Wis.
Grell, E.H. Oak Ridge, Tenn.
Grell, R.F. Oak Ridge, Tenn.
Greuter, M. Switzerland, Zürich
Griech, H.A. Philadelphia, Pa.
Griffen, A.B. Bar Harbor, Maine.
Grimes, J. DeKalb, Ill.
Grisseau, C.R. Columbus, Ohio
Grun, P. University Park, Pa.
Gruneberg, H. Gr. Britain, London
Grzegorzewska, M.D. Brazil, São Paulo
Guerrier, P. France, Lyon
Guest, W.C. University, Ala.
Gugler, D.H. Duarte, Calif.
Guglielmi, A. Italy, Pavia
Guillaumin, M. France, Paris
Gunson, M.M. Australia, Melbourne

Haas, F. Houston, Texas
Hadorn, E. Switzerland, Zürich
Hagens, H.W. Germany, Heidelberg
Haldane, J.B.S. India, Calcutta
Halfer, C. Germany, Berlin-Dahlem
Halfer, C. Italy, Milano
Hall, J. Bloomington, Ind.
Halpern, L. Fayetteville
Hamilton, J. Eugene, Ore.
Hanks, G.D. Eugene, Ore.
Hanly, E.W. Austin, Texas
Hannah-Alava, A. Finland, Turku
Hanson, A. Logan, Utah
Hardin, R.T. Lafayette, Ind.
Harlock, R. Australia, Brisbane
Harrington, D. Tucson, Ariz.
Harrison, P.A. Lexington, Ky.
Hartman, I.J. S. Africa, Johannesburg
Hartshorne, J.N. Gr. Britain, Manchester
Hauser, A.T. Philadelphia, Pa.
Haverland, L.H. Ames, Iowa
Hayman, D.L. Australia, Adelaide
Heed, W. Tucson, Ariz.
Heinonen, P. Finland, Turku
Heinsoo, M. Eugene, Ore.
Helling, R.B. Pittsburgh, Pa.
Henderson, T.C. Pasadena, Calif.
Henke, H. Germany, Hamburg-Eppendorf
Henning, M. Bloomington, Ind.
Henningsen, K. Denmark, Copenhagen
Herskowitz, I.H. St. Louis, Mo.
Hess, O. Germany, Tübingen
Heuts, M.J. Belgium, Louvain
Hexter, W.M. Amherst, Mass.
Higgins, W. Bar Harbor, Maine
Hildreth, P. Berkeley, Calif.
Hillman, R. Philadelphia, Pa.
Hinton, C.W. Athens, Georgia
Hiraga, S. Japan, Osaka
Hiraizumi, Y. Japan, Misima
Hiroyoshi, T. Japan, Osaka
Hirsh, D.I. Portland, Ore
Hochman, B. Oak Ridge, Tenn.
Hoenigsberg, H.F. Italy, Milano
Höhne, G. Germany, Hamburg-Eppendorf
Hollander, W.F. Ames, Iowa
Holler, A. Philadelphia, Pa.
Hollingsworth, M.J. Ghana, Legon
Horn, S. Berkeley, Calif.
Hotz, G. Germany, Heidelberg
House, M. Columbus, Ohio
House, V.L. Columbus, Ohio
Howe, M.L. Lawrence, Kan.
Hope, L. Gr. Britain, Chalfont St. Giles
Hubby, J.L. Chicago, Ill.
Hughes, R.D. Richmond, Va.
Hungerford, D.A. Philadelphia, Pa.
Hunter, A.S. Colombia, Bogotá
Hurvitz, D. Israel, Jerusalem

Ichioka, S. Japan, Osaka
Ikeda, H. Japan, Tokyo
Imai, Y. Japan, Misima
Imaizumi, T. Japan, Kyoto
Inagaki, E. Japan, Chiba
Ingrahm, R. Berkeley, Calif.
Inouye, I. Japan, Okamoto

Ives, P.T. Amherst, Mass.
Iyama, S. Japan, Misima

Jackson, D.M. Chapel Hill, N.Carolina
Jacobs, M.E. Bethany, W.Virginia
Jais, J.A. Chicago, Ill.
Janke, H. Salt Lake City, Utah
Jansson, A. Sweden, Uppsala
Jayakar, S.D. India, Calcutta
Jempson, J. Gr. Britain, Harwell
Johansson, K. Sweden, Uppsala
Johnsen, R. Eugene, Ore.
Johnson, G.R. Salt Lake City, Utah
Johnson, W.W. Minneapolis, Minn.
Jones, C. Bloomington, Ind.
Jonsson, U. Cambridge, Mass.
Judd, B.H. Austin, Texas
Juvonen, V. Finland, Turku

Kaji, S. Japan, Okamoto
Kaminishi, H. Japan, Mitaka
Kanehisa, T. Japan, Kobe
Kaneko, A. Japan, Sapporo
Kang, B.J. Korea, Seoul
Kang, Y.S. Korea, Seoul
Kaplan, W.D. Duarte, Calif.
Karlik, A. Austria, Vienna
Kasai, I. Japan, Chiba
Kashiwabara, T. Berkeley, Calif.
Kato, M. Japan, Kyoto
Kato, S. Los Angeles, Calif.
Kaufman, A.F. Corvallis, Ore.
Kaufman, S. Switzerland, Zürich
Kaufmann, B.P. Cold Spring Harbor, N.Y.
Kawabe, M. Japan, Kobe
Kaye, R.M. Rochester, N.Y.
Keller, E.C.,Jr. University Park, Pa.
Kelsall, P.J. Gr. Britain, Edinburgh
Kernaghan, R.P. Cold Spring Harbor, N.Y.
Kidwell, J.F. Ames, Iowa
Kiil, V. Norway, Blindern
Kikkawa, H. Japan, Osaka
Kim, D.U. Korea, Kwangju
Kim, K.W. Korea, Kwangju
Kim, M.C. Korea, Seoul
Kim, O.S. Korea, Kongju
Kim, T.S. Korea, Seoul
Kimura, M. Japan, Misima
Kindernay, B.L. Johnson City, Tenn.
King, J. Berkeley, Calif.
King, R.C. Evanston, Ill.
Kirschbaum, W.F. Argentina, Buenos Aires
Kitagawa, O. Japan, Tokyo
Kitazume, Y. Japan, Kobe
Kheiralla, A. Gr. Britain, Nottingham
Knight, G.R. Gr. Britain, Edinburgh
Khishin, A.F. United Arab Rep., Assuit
Kobayashi, M. Salt Lake City, Utah
Koch, E.A. Evanston, Ill.
Köhnlein, W. Germany, Heidelberg
Kolb, J.A. Salt Lake City, Utah
Kolenkine, X. France, Lyon
Konarski, M. Philadelphia, Pa.
Kondo, K. Japan, Anzyo-She
Koref-Santibañez, S. Chile, Santiago
Koske-Westphal, Germany, Hamburg
Kosswig, C. Germany, Hamburg
Kotte, J. Germany, Berlin-Dahlem
Krause, E. Lafayette, Ind.
Krauss, M. Cold Spring Harbor, N.Y.
Krawinkel, M.E. Lafayette, Ind.
Krebs, J.J. St. Louis, Mo.
Krivshenko, E. Rochester, N.Y.
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Kromm, N. Germany, Berlin-Dahlem
Krupka, J.W. Lincoln, Neb.
Künkel, H.A. Germany, Hamburg-Eppendorf
Kunze-Mühl, E. Austria, Vienna
Kuroda, Y. Japan, Osaka
Kurokawa, H. Japan, Tokyo
Kvelland, I. Norway, Blindern
Kyle, W.H. Lafayette, Ind.

Lafferty, B.E. Lexington, Ky.
Lamerton, M. Gr. Britain, Harwell
Landy, R. DeKalb, Ill.
Lane, E. Eugene, Ore.
Langer, B. Pittsburgh, Pa.
Laufer, H. Baltimore, Md.
Laugé, G. France, Gif-sur-Yvette
Laureys, M.J. Belgium, Louvain
Laves, B.A. Chicago, Ill.
Lawrence, M.J. Gr. Britain, Birmingham
Lawson, J.E. Johnson City, Tenn.
Leboulleux, P. France, Gif-sur-Yvette
Lederman-Klein, A. Israel, Jerusalem
Lee, H.Y. Korea, Seoul
Lee, T.J. Korea, Kongju
Lefevre, G. Cambridge, Mass.
Legay, J.M. France, Lyon
Lehtonen, A. Finland, Turku
Leon, W.N. Argentina, Buenos Aires
Leppin, H. Germany, Berlin-Buch
Lesher, S.W. Argonne, Ill.
Lestrange, M. France, Gif-sur-Yvette
Leventhal, E. New Haven, Conn.
Levine, O. Israel, Jerusalem
Levine, R.P. Cambridge, Mass.
Levins, R. Rochester, N.Y.
Lewis, E.B. Pasadena, Calif.
Lewis, H.S. Cambridge, Mass.
Lewis, H.W. Cambridge, Mass.
Lewontin, R.C. Rochester, N.Y.
L'Héritier, Ph. France, Gif-sur-Yvette
Lichtwardt, E. Lawrence, Kan.
Lindqvist, B. Sweden, Stockholm
Lindsley, D.L. Brazil, São Paulo
Lints, F.A. Belgium, Louvain
Lippman, E. Philadelphia, Pa.
Locatelli, F. Italy, Milano
Louis, M. France, Gif-sur-Yvette
Lovellette, E.J. San Diego, Calif.
Lucchesi, J. Berkeley, Calif.
Luce, W.M. Urbana, Ill.
Luchowski, E. Buffalo, N.Y.

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Ludwig, H. Germany, Heidelberg
Lüers, H. Germany, Berlin-Dahlem
Lüers, T. Germany, Berlin-Dahlem
Luerson, C. Austin, Texas
Lund, B. Ithaca, N.Y.
Lund, B. Sweden, Uppsala
Lund, D.E. Lincoln, Neb.
Lüning, K.G. Sweden, Stockholm

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McCracken, M. Eugene, Ore.
McCrady, W.B. Lincoln, Neb.
McCullough, M. Los Angeles, Calif.
McDowell, S.B. Newark, N.J.
McHale, F. Ithaca, N.Y.
McKean, H.E. Lafayette, Ind.
McKinley, K. Houston, Texas
McNew, R.A. Baton Rouge, La.
McSheehy, T.W. Gr. Britain, Harwell
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Magalhães, L.E. Brazil, São Paulo
Magni, G.E. Italy, Pavia
Mahowald, A.P. Baltimore, Md.
Mainx, F. Austria, Vienna
Makino, S. Japan, Sapporo
Mallah, G.S. United Arab Rep., Assuit
Mange, E.J. Madison, Wis.
Markowitz, E. Pasadena, Calif.
Martin, M. Newark, N.J.
Martin, W.J. Salt Lake City, Utah
Marzluf, G.A. Baltimore, Md.
Massamállo, L. Philadelphia, Pa.
Masterson, J. Eugene, Ore.
Masuda, H. Japan, Misima
Mather, K. Gr. Britain, Birmingham
Mather, W.B. Australia, Brisbane
Matsumoto, R. Philadelphia, Pa.
Maurer, P.R. Philadelphia, Pa.
Mayeda, K. Salt Lake City, Utah
Mayhew, S. Los Angeles, Calif.
Maynard Smith, J. Gr. Britain, London
Mayo, M.J. Australia, Adelaide
Mazar-Barnet, B. Argentina, Buenos Aires
Mead, C.G. East Lansing, Mich.
Merckx, F.E. Belgium, Louvain
Megna, F. Italy, Napoli
Meloni, M.L. Philadelphia, Pa.
Melou, J.P. France, Paris
Merrell, D.J. Minneapolis, Minn.
Meyer, H.U. Bloomington, Ind.
Mickey, G.H. Ridgefield, Conn.
Milkman, R.D. Syracuse, N.Y.
Miller, D.D. Lincoln, Neb.
Miller, M. Gr.Britain, Chalfont St.Giles
Millhouse, E.W. Urbana, Ill.
Miltenburger, H. Germany, Heidelberg
Minamori, S. Japan, Hiroshima
Miralles, L. Spain, Madrid
Misra, R.K. Gr. Britain, Edinburgh
Mitchell, H.K. Pasadena, Calif.
Mitra, J. India, Calcutta
Mittler, S. DeKalb, Ill.
Miyoshi, Y. Japan, Kyoto
Mohler, J.D. Corvallis, Ore.
Mohn, N. Pittsburgh, Pa.
Mohr, O.L. Norway, Blindern
Momma, E. Japan, Sapporo
Monclús, M. Spain Barcelona
Monkman, M. DeKalb, Ill.
Montalenti, G. Italy, Napoli
Montelius, I. Sweden, Stockholm
Montgomery, D. Eugene, Ore.
Montgomery, E. DeKalb, Ill.
Montgomery, R. Houston, Texas
Moore, I. Los Angeles, Calif.
Moree, R. Pullman, Wash.
Morey, M. Spain, Madrid
Morio, H. France, Gif-sur-Yvette
Moriwaki, D. Japan, Tokyo
Motobu, H. Japan, Kyoto
Mourad, A.M. United Arab Rep., Alexandria
Mouravieff, A.N. Belgium, Louvain
Moyer, S.E. Minneapolis, Minn.
Moynehan, J.P. Lafayette, Ind.
Mukai, T. Japan, Misima
Mukherjee, A. Berkeley, Calif.
Mukherjee, M. India, Calcutta
Müller, A. Germany, Heidelberg
Muller, H.J. Bloomington, Ind.
Muller, M. Switzerland, Zürich
Muñoz, E.R. Argentina, Buenos Aires
Munz, P. Switzerland, Zürich
Myers, O.,Jr. Ithaca, N.Y.
Myers, T.L. East Lansing, Mich.

Nadal, A. Spain, Barcelona
Nakamura, K. Japan, Kyoto
Nakamura, K. Japan, Misima
Nakao, Y. Japan, Chiba
Narayana, R. India, Madras
Narise, T. Japan, Misima
Nash, D. Gr. Britain, Cambridge
Nash, D.J. University Park, Pa.
Nawa, S. Japan, Misima
Neeley, J.R. Corvallis, Ore.
Neubert, J. Austria, Vienna
Neulat, M.M. France, Lyon
Newball, S. Colombia, Bogotá
Newby, W.W. Salt Lake City, Utah
Nicklas, R.B. New Haven, Conn.
Nicoletti, B. Oak Ridge, Tenn.
Nieuwenhuyzen, A. Netherlands, Leiden
Nigon, V. France, Lyon
Nobuki, R. Japan, Osaka
Nolte, D.J. South Africa, Johannesburg
Nonami, K. Japan, Misima
Nothel, H. Germany, Berlin-Dahlem
Nonnenmacher, J. France, Lyon
Nothiger, R. Switzerland, Zürich
Novitski, E. Eugene, Ore.
Nozawa, K. Japan, Anzyo-Shi

Ogaki, M. Japan, Sakai
Ogita, Z. Japan, Osaka
Oh, T.S. Korea, Seoul

Ohanessian-Guillemain,A.France,Gif-sur-Yv.
Ohba, S. Japan, Tokyo
Ohlendorff, H. Sweden, Uppsala
Ohnishi, E. Japan, Tokyo
Okada, T. Japan, Tokyo
Oksala, T.A. Finland, Turku
Okuda, C. Japan, Kyoto
Oliver, C.P. Austin, Texas
Olivieri, G. Italy, Napoli
O'Neal, L.L. Newark , N.J.
Orillard, C. France, Paris
Ortiz, E. Spain, Madrid
Oshima, C. Japan, Misima
Oster, I.I. Philadelphia, Pa.
Ostertag, W. Bloomington, Ind.
Ota, N. Japan, Anzyo-Shi

Paik, Y.K. Korea, Seoul
Paolini, F.A. Switzerland, Zürich
Park, M.S. Korea, Kwangju
Parker, D.R. Riverside, Calif.
Parker, N.R. Austin, Texas
Parsons, P.A. Gr. Britain, Cambridge
Parzen, S.D. East Lansing, Mich.
Passano, K.N. New Haven, Conn.
Pasternak, L. Germany, Berlin-Dahlem
Paterson, H.E. S. Africa, Johannesburg
Patterson, D.L. Eugene, Ore.
Patterson, J.T. Austin, Texas
Pavan, C. Brazil, São Paulo
Paz, B.C. Argentina, Buenos Aires
Peat, W.E. Gr. Britain, Nottingham
Pelekanos, M. Gr. Britain, Cambridge
Pellicer, M. Chile, Santiago
Perdrix, S. France, Lyon
Perras, J.P. East Lansing, Mich.
Perrayon, R. France, Lyon
Perry, M. Gr. Britain, Edinburgh
Perry, T.L. Johnson City, Tenn.
Peruzzi, T. Ridgefield, Conn.
Peschiera, G. Italy, Pavia
Petäjä, T. Finland, Turku
Peters, W. Chicago, Ill.
Petit, C. France, Paris
Phillips, C. Los Angeles, Calif.
Pilgrim, I.H. Salt Lake City, Utah
Pinto, S.C. Brazil, São Paulo
Pipkin, S.B. Panama, Panama
Pistey, W.R. Ridgefield, Conn.
Piternick, L.K. Berkeley, Calif.
Piva, A. France, Gif-sur-Yvette
Plaine, H.L. Columbus, Ohio
Plough, H.H. Amherst, Mass.
Plus, N. France, Gif-sur-Yvette
Pollard, L.D. Tallahassee, Fla.
Pollock, R. Norman, Okla.
Polzin, W. Germany, Berlin-Dahlem
Pons, J. Spain, Barcelona
Pontecorvo, G. Gr. Britain, Glasgow
Pooley, E.H. Philadelphia, Pa.
Poulson, D.F. New Haven, Conn.
Pozzi, L. Italy, Milano
Prevosti, A. Spain, Barcelona
Pringle, C.R. Gr. Britain, Aberdeen
Prokop, B. Cold Spring Harbor, N.Y.
Proust, J. France, Gif-sur-Yvette
Prout, T. Riverside, Calif.
Ptashne, M. Portland, Ore.
Purdom, C.E. Gr. Britain, Harwell
Puro, J. Finland, Turku

Queiroz, J. France, Gif-sur-Yvette
Querner, W. Germany, Mariensee

Rai, K.S. Notre Dame, Indiana
Ramel, C. Sweden, Stockholm
Ramírez, P. Spain, Madrid
Rapaport, I. Madison, Wis.
Rapaport, S. Israel, Jerusalem
Rasmuson, B. Sweden, Uppsala
Rasmuson, M. Sweden, Uppsala
Rasmussen, I.E. Italy, Pavia
Ratty, F.J. San Diego, Calif.
Ray-Chaudhuri, S.P. India, Calcutta
Redfield, H. Philadelphia, Pa.
Reeve, E.C.R. Gr. Britain, Edinburgh
Rein, A. Portland, Ore.
Rekemeyer, B. Oak Ridge, Tenn.
Rendel, J.M. Australia, Sydney
Rerolle, M. France, Lyon
Rezzonico, R.G. Italy, Milano
Richter, J. South Africa, Johannesburg
Ridgeway, T.N. Rochester, N.Y.
Riles, L.S. St. Louis, Mo.
Rinehart, R.R. Austin, Texas
Rinkel, R. Lawrence, Kan.
Ritossa, F. Italy, Pavia
Ritte, U. Israel, Jerusalem
Ritterhoff, R.K. Baltimore, Md.
Rizki, R.M. Portland, Ore.
Rizki, T.M. Portland, Ore.
Roberts, P.A. Chicago, Ill.
Robertson, A. Gr. Britain, Edinburgh
Robertson, F.W. Gr. Britain, Edinburgh
Roderick, T.H. Bar Harbor, Maine
Rodgers, E. Madison, Wis.
Rodríguez, C. Spain, Madrid
Rohlf, F.J. Lawrence, Kan.
Rohrborn, G. Germany, Berlin-Dahlem
Ronen, A. Israel, Jerusalem
Roper, J.A. Gr. Britain, Sheffield
Rosin, S. Switzerland, Bern
Rosner, J.L. New Haven, Conn.
Rothe, H. Los Angeles, Calif.
Royes, V. Gr. Britain, Edinburgh
Ruderer, E. Austria, Vienna
Rudkin, G.T. Philadelphia, Pa.
Rudolph, E. Germany, Berlin-Dahlem
Rümke, C.L. Netherlands, Utrecht
Russell, P. Amherst, Mass.
Russell, J. Tucson, Ariz.
Ruttner, F. Austria, Vienna

Saarinen, L. Portland, Ore.
Sakaguchi, B. New Haven, Conn.
Sakai, K. Japan, Misima

Sakitey, A.S. Ghana, Legon
Salivar, W. Argonne, Ill.
Samuelson, K. Syracuse, N.Y.
Sandler, L. Madison, Wis.
Sang, J.H. Gr. Britain, Edinburgh
Sarkar, S.K. India, Calcutta
Savage, E. Alliance, Ohio
Savhagen, R. Sweden, Stockholm
Savolainen, S. Finland, Turku
Sayers, R. Ithaca, N.Y.
Scossiroli, R.E. Italy, Pavia
Schaible, R.H. Ames, Iowa
Schalet, A. Cold Spring Harbor, N.Y.
Scharloo, W. Netherlands, Leiden
Schepers, A. Netherlands, Leiden
Scheren, A.M. Philadelphia, Pa.
Schlager, G. Lawrence, Kan.
Schlapfer, T. Switzerland, Zürich
Schmid, W. Austin, Texas
Schneider, I.P. Chicago, Ill.
Schnitter, M. Switzerland, Zürich
Schouten, S.C.M. Netherlands, Utrecht
Schubert, G. Germany, Hamburg-Eppendorf
Schuellein, R.J. Dayton, Ohio
Schultz, J. Philadelphia, Pa.
Schwarz, R. Philadelphia, Pa.
Schweda, M. Lawrence, Kan.
Schwinck, I. Germany, Mariensee
Scriba, M. Germany, Marburg
Seaton, R.K. East Lansing, Mich.
Seecof, R.L. Pasadena, Calif.
Seidel, F. Germany, Marburg
Seidel, S. Germany, Tübingen
Seiger, M.B. Canada, Toronto
Seki, T. Japan, Osaka
Sen, B.K. Gr. Britain, Edinburgh
Seto, F. Berea, Ky.
Sgorbati, G. Italy, Milano
Shahaph, J. Israel, Jerusalem
Shapard, P.B. Washington, D.C.
Shapiro, N. Lafayette, Ind.
Sheldon, B.L. Australia, Sydney
Sheldon, B.L. Pasadena, Calif.
Sherwood, E. Berkeley, Calif.
Shideler, D. Lafayette, Ind.
Shima, T. Japan, Sapporo
Shiomi, M. Japan, Chiba
Shiomi, T. Japan, Chiba
Shoji, T. Japan, Mitaka
Shoopman, J. Berea, Ky.
Sick, K. Denmark, Copenhagen
Sigot, A. France, Strasbourg
Sims, M.M. Eugene, Ore.
Singer, K.M. Cambridge, Mass.
Singh, I.P. East Lansing, Mich.
Singleton, J.R. Lafayette, Ind.
Sinoto, Y. Japan, Mitaka
Sironi, G.P. Italy, Milano
Slatis, H.M. Argonne, Ill.
Slizynski, B.M. Gr. Britain, Edinburgh
Slizynska, H. Gr. Britain, Edinburgh
So, W.Y. Korea, Seoul
Sokal, R.R. Lawrence, Kan.
Sokoloff, A. Los Angeles, Calif.
Solana, I. Spain, Madrid
Solima, A. Italy, Napoli
Sollunn, F.J. Norway, Blindern
Sondhi, K.C. Gr. Britain, London
Sonnenblick, B.P. Newark, N.J.
Sorensen, W.K. Logan, Utah
Southin, J. Los Angeles, Calif.
Souza, H.M.L. Brazil, São Paulo
Sperlich, D. Austria, Vienna
Spickett, S.G. Gr. Britain, Cambridge
Spieler, R.A. Chicago, Ill.
Spiess, L.D. Pittsburgh, Pa.
Spieth, H.T. Riverside, Calif.
Spofford, J.B. Chicago, Ill.
Springer, R. Austria, Vienna
Spurway, H. India, Calcutta
Spurway, R. Australia, Brisbane
Stern, C. Berkeley, Calif.
Stadler, J. Ames, Iowa
Stalker, H.D. St. Louis, Mo.
Stanich, G.J. East Lansing, Mich.
Steffensen, D. Upton, N.Y.
Steinberg, A.G. Cleveland, Ohio
Steinitz, J. Salt Lake City, Utah
Stevenson, R. Johnson City, Tenn.
Stiers, R.O. Belgium, Louvain
Stone, L.E. Lincoln, Neb.
Stone, W.S. Austin, Texas
Stough, P. DeKalb, Ill.
Strangio, V.A. Australia, Melbourne
Stratopoulos, G. Salt Lake City, Utah
Strømnaes, Ø. Norway, Blindern
Struck, E. Germany, Berlin-Dahlem
Sturtevant, A.H. Pasadena, Calif.
Sturtevant, J. Ames, Iowa
Suguna, S.G. India, Madras
Sulerud, R.L. Lincoln, Neb.
Sullivan, R.L. Lawrence, Kan.
Sung, K.C. Korea, Seoul
Suzuki, D.T. Chicago, Ill.
Svensson, M. Sweden, Uppsala
Sweeney, E.A. East Lansing, Mich.
Sweet, E.E. Pittsburgh, Pa.
Sweron, M. Gr.Britain, Chalfont St.Giles

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Taira, T. Japan, Misima
Takada, H. Austin, Texas
Takatsu, Y. Brazil, São Paulo
Takaya, H. Japan, Okamoto
Talsna, J. Cold Spring Harbor, N.Y.
Tanaka, E. Urbana, Ill.
Tano, S. Lafayette, Ind.
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Tassencourt, France, Gif-sur-Yvette
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Taylor, W.P. San Diego, Calif.
Teissier, G. France, Gif-sur-Yvette
Teissier, G. France, Paris
Tellegen, A. Minneapolis, Minn.

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Thomas, C. Salt Lake City, Utah
Thomas, S. Bloomington, Ind.
Thomassen, I.L. Norway, Blindern
Thompson, J.A. Australia, Melbourne
Thompson, P.E. Oak Ridge, Tenn.
Thornley, P.A. Athens, Georgia
Throckmorton, L. Riverside, Calif.
Tiffany, B. Amherst, Mass.
Tinderholt, V. Duarte, Calif.
Tinderholt, V.E. Los Angeles, Calif.
Tobari, I. Japan Tokyo
Tobari, Y. Japan, Tokyo
Toledo, J.S. Brazil, São Paulo
Toledo, S.A. Brazil, São Paulo
Tomita, T. Japan, Anzyo-Shi
Torroja, E. Spain, Madrid
Townsend, J.I. Richmond, Va.
Toyofuku, Y. Japan, Sapporo
Traut, H. Germany, Heidelberg
Travaglini, E.C. Philadelphia, Pa.
Treanor, K. Buffalo, N.Y.
Trosko, J.E. East Lansing, Mich.
Trout, W.E. Bloomington, Ind.
Tsukamoto, H. Japan, Misima
Tsukamoto, M. Japan, Osaka
Tuinstra, E.J. Netherlands, Utrecht
Twesten, M.A. Philadelphia, Pa.

Ulrich, H. Switzerland, Zürich
Ursprung, H. Switzerland, Zürich
Usciati, J. France, Paris

Valencia, J.I. Argentina, Buenos Aires
Valencia, R.M. Argentina, Buenos Aires
Vanoueck, E.D. Ithaca, N.Y.
Vate, C.V.D. Netherlands, Utrecht
Vigier, Ph. France, Gif-sur-Yvette
Vinuela, E. Spain, Madrid
Virzo, A. Italy, Napoli
Vloeberghs, J.V. Belgium, Louvain
Volkart, H. Switzerland, Bern
Volkers, W. Netherlands, Leiden
von Borstel, R.C. Oak Ridge, Tenn.
Von Halle, E.S. Oak Ridge, Tenn.
Waddington, C.H. Gr. Britain, Edinburgh
Wagner, R.P. Austin, Texas
Wagoner, D.E. Bloomington, Ind.
Wahrman, J. Israel, Jerusalem
Wajntal, A. Brazil, São Paulo
Walker, I. Switzerland, Zürich
Wallace, B. Brazil, São Paulo
Wallbrunn, H.M. Gainesville, Fla.
Wang, S.C.C. Columbus, Ohio
Ward, C.L. Durham, N.Carolina
Warfield, S.L. Baltimore, Md.
Wasserman, M. Australia, Melbourne
Watanabe, H. Japan, Osaka
Wattiaux, J.M. Belgium, Louvain
Weingart, E.A. Cold Spring Harbor, N.Y.
Welch, R.M. Austin, Texas
Weinmann, H. Switzerland, Zürich
Weiss, J.R. Wellesley, Mass.
Welshons, W.J. Oak Ridge, Tenn.
Westergaard, M. Denmark, Copenhagen
Wette, R. Germany, Heidelberg
Wharton, M. St. Louis, Mo.
Wheeler, M.R. Austin, Texas
White, E. Houston, Texas
Whitsel, B. Los Angeles, Calif.
Whittinghill, M. Chapel Hill, N.Carolina
Whittington, W.J. Gr. Britain, Nottingham
Whurr, A. Gr. Britain, Chalfont St. Giles
Wilhelm, B. Philadelphia, Pa.
Wilkerson, R. Oak Ridge, Tenn.
Williams, T. St. Louis, Mo.
Wilson, F.D. Austin, Texas
Wilson, L.P. Wellesley, Mass.
Williamson, D.L. Salt Lake City, Utah
Winterfeld, G. Germany, Berlin-Dahlem
Wolf, E. Germany, Berlin-Dahlem
Wolf, J. Newark, N.J.
Woolf, C.M. Salt Lake City, Utah
Wolff, M. Eugene, Ore.
Woods, P.S. Upton, N.Y.
Wolstenholme, D.R. Gr. Britain, Cambridge
Wright, E.Y. Baltimore, Md.
Wright, T.R.F. Baltimore, Md.
Wui, I.S. Korea, Kwangju
Würgler, F.E. Switzerland, Zürich
Wyllie, G.A. Lafayette, Ind.

Yamada, Y. Japan, Misima
Yanders, A.F. East Lansing, Mich
Yarkoni-Freund, O. Israel, Jerusalem
Yasuda, N. Japan, Chiba
Yeatts, V.D. Columbus, Ohio
Yoon, S.B. East Lansing, Mich.
Yoshida, Y.H. Japan, Tokyo
Yost, H.T. Jr. Amherst, Mass.
Ytterborn, K. Sweden, Stockholm

Zebe, E. Germany, Heidelberg
Zimmer, K.G. Germany, Heidelberg
Zimmering, S. Bloomington, Ind.
Zimmermann, W. Germany, Mariensee
Zürcher, C. Switzerland, Zürich

(continued from page 8)

much less than that in the immediately preceeding issues, although it appears at first sight to be somewhat smaller.

The photo-offset process has made possible certain other improvements. The prohibition against line drawings is now removed. Such drawings should be on a separate sheet of paper, in black ink, about ten per cent larger in both dimensions than it should appear when reproduced. Equally advantageous is the possibility of reproducing photographs (see page 118). This, however, does involve additional expense which will have to be borne by the contributor. A half-tone reproduction 3 x 4 inches (or smaller) will cost $5.00 and one larger than this size, up to 4 x 6 inches will cost $7.50. At the present time we do not contemplate including any size larger than 4 x 6 inches.

Publishability of notes: The question of whether DIS is now a bona fide publication or not, or should be made one if it isn't, was discussed at length by the Advisory Board who expressed the same wide range of sentiments that workers in the field themselves must feel. The problem was resolved by deciding to maintain the status quo, with the proviso that when notes were of such a nature that the Editor felt that they might better be presented in a publication rather than DIS, he should feel free to reject such contributions. (It is the opinion of the Editor that as long as no publication is willing to accept the kind of terse technical notes as are now regularly submitted to DIS, it must continue to accept them.) The custom, in the past, of designating by an asterisk a note that may be quoted without permission, or of giving blanket prior approval to all notes, may have the disadvantage of discouraging those short, chatty, informal informational notes that may later prove to be in error in some respect as additional data accumulate. The Editor would like to suggest another possibility: that at the time that contributions are sent in, each worker indicate which of his notes appearing in earlier issues may be quoted. Such a list in DIS from time to time would eliminate the necessity of direct contact with the author of each note each time it is referred to. The next time calls for material are sent to Drosophilists, a request will be made for this information.

Editing: Changes in copy have been made very rarely and only in those cases where it appeared unquestionable that a change would make a vast improvement. More important is the question of acceptance or rejection of notes. My attitude is this: a short informal note about work in progress or covering some minor observations will generally be acceptable without question, but a detailed account of a completed experiment written in such a way as to imply a small publication should be subjected to the same scrutiny, possibly by reviewers, that it would get if submitted to a bona fide publication. Several notes intended for this issue, falling in the latter category, have been so treated, and, in a few cases, have been returned to the author.

Volume of the contributions: The number of notes submitted has risen markedly. All those received could not be included in the present issue; those originating in the United States which arrived after the deadline are being held for the next issue (DIS-35) which, it is hoped, will appear in about six months or less. No call will be sent out for DIS-35 and contributions are welcome at any time. That issue will include the Bibliography, which has been omitted from this one and will not include a Directory. The distribution of DIS-35 will be the same as for DIS-34.

There are several reasons for this decision. One is that a single issue per year is not adequate to take care of all the material received (although it would appear at the present time that two issues per year would be more than is required). The second major reason for this suggestion is that it appears desirable to avoid the intense activity lasting now for just the few months that DIS is typed, edited and printed, and to spread the work involved in a more relaxed manner over a longer period of time.

Sale of copies: Any interested person may now receive a copy of DIS for the sum of $2.00. (Please make checks payable to DIS.) In order to minimize the paper work and mailing costs involved in this distribution, it is hoped that workers with research funds against which such payment might readily be charged will anticipate their future DIS needs for assistants and graduate students by ordering an adequate supply of copies at one time. The sums received will help support future issues of DIS (NSF funds are now not completely adequate for this purpose) and will be used also to cover the cost of condensations of the research and technical notes of issues prior to number 34.